Livingstones' Medical Handbooks.

SYMINGTON'S TOPOGRAPHICAL ANATOMY of the CHILD,	£2, 2/0
EDINBURGH MEDICAL SCHOOL CALENDAR, by post,	1/8
GREVILLE'S CHEMISTRY, with Tables, *2nd Edition,*	6/0
MILNE'S MIDWIFERY, *2nd Edition,*	12/6
Do. MANAGEMENT OF CHILDREN,	2/6
HUSBAND'S (Aubrey) FORENSIC MEDICINE and PUBLIC HEALTH, *4th Edition,*	10/6
Do. PRACTICE OF MEDICINE, *4th Edition.*	In the Press.
Do. POCKET PRESCRIBER,	1/0
Do. SANITARY LAW,	3/6
Do. EXAMINATION OF THE URINE,	1/0
HUGHES' MOTOR NERVES,	1/0
CROOM'S MINOR GYNECOLOGY, *2nd Edition,*	6/0
CRAIG'S MATERIA MEDICA, *5th Edition,*	6/0
Do. POSOLOGICAL TABLES,	1/0
WALLER'S (Bryan) INTERSTITIAL NEPHRITIS,	4/6
M'LACHLAN'S ANATOMY OF SURGERY, *2nd Edition,*	10/6
SUTHERLAND—HOSPITALS and their Construction,	3/0
WHITAKER—THE BRAIN AND SPINAL CORD, with Plates,	4/6
WEST'S VERTEBRATE ZOOLOGY,	1/6
CASE-TAKING BOOK for Hospitals,	0/6
MATERIA MEDICA CARDS—Organic and Inorganic (Two Sets), *Catechism Series,*	Per set, net, 2/6
PATHOLOGY CARDS—General and Special (Two Sets), *or in Note Book form, Catechism Series*	Per set, net, 2/6
J	each 2/0
	2/0
	the Press.

22500447380

Med
K26141

J. B. H. Beatty
St Bartholomew's Hospital,
Smithfield, E.C.
6 · 4 · 88.

THE STUDENT'S HAND-BOOK

OF THE

PRACTICE OF MEDICINE

7329

THE STUDENT'S HAND-BOOK
OF THE
PRACTICE OF MEDICINE

DESIGNED FOR THE USE OF STUDENTS PREPARING
FOR EXAMINATION.

By H. AUBREY HUSBAND, M.B., C.M.,
B.Sc., F.R.C.S.E., M.R.C.S., L.S.A.,
LATE LECTURER ON MEDICAL JURISPRUDENCE AND PUBLIC HEALTH IN THE
EXTRA-ACADEMICAL SCHOOL, EDINBURGH.
Member of the General Council of the University of Edinburgh; and
Extraordinary Member of the Royal Medical Society,
Edinburgh.
Author of 'Student's Hand-Book of Forensic Medicine and Public Health,'
'Sanitary Law,' 'Medical and Surgical Examination Questions,'
'The Urine in Health and in Disease,' etc.

FOURTH EDITION, REVISED AND ENLARGED
With Illustrations and Tables.

EDINBURGH:
E. & S. LIVINGSTONE, 15 TEVIOT PLACE.
1888.

10442

PRINTED BY
E. & S. LIVINGSTONE
4 MELBOURNE PLACE
EDINBURGH.

LONDON: SIMPKIN, MARSHALL, AND CO.;
HAMILTON, ADAMS, AND CO.; BAILLIÈRE, TINDALL, AND COX.
GLASGOW: SIME; STENHOUSE.
DUBLIN: FANNIN AND CO.; GILL AND SON.
MANCHESTER: J. E. CORNISH.

29899 936

WELLCOME INSTITUTE
LIBRARY

Coll.	welMOmec
Call	
No.	WB

PREFACE TO THE FOURTH EDITION.

In this Edition the whole volume has been carefully revised, considerable additions being made to those portions on the Nervous and Circulatory Systems. New Tables in aid to Diagnosis have been added, together with several Illustrations.

The recent edition of the nomenclature of Disease, put forth by the London College of Physicians, has been adopted in this Edition.

CONTENTS.

SECTION I.

GENERAL PATHOLOGY.

PAGE
Hypertrophy, ... 3
Atrophy, ... 4
Hyperæmia, ... 5
Hæmorrhage, ... 9
Dropsy, ... 10
Inflammation, ... 12
Degeneration, ... 15
Fatty Degeneration, ... 16
Mucoid Degeneration, ... 18
Colloid Degeneration, ... 19
Lardaceous Degeneration, ... 19
Calcareous Infiltration, ... 20
Pigmentary Infiltration, ... 21
Fatty Infiltration, ... 21

SECTION II.

GENERAL DISEASES.

Group *A.*—Diseases dependent on Morbid Poisons.

Sub-Group (a).—Pyrexiæ—Fevers.

General Treatment of Fevers, ... 28
Febricula, ... 32

PAGE
Relapsing Fever, ... 35
Typhus Fever, ... 41
Typhoid Fever 48
Infantile Remittent Fever, ... 58
Cerebro-Spinal Meningitis, ... 61
Yellow Fever, ... 63

Variola, or Small-Pox, ... 70
Varioloid, ... 77
Vaccinia, or Cow-Pox, ... 78
Varicella, or Chicken-Pox, ... 80
Scarlatina, or Scarlet Fever, ... 81
Dengue, ... 87
Morbilli, Rubeola, or Measles, ... 88
Rötheln, Rubeola, or German Measles, . . 90

Plague, ... 93
Epidemic Cholera, ... 93
Influenza, ... 103
Diphtheria, ... 104
Whooping-Cough, ... 110
Parotitis, ... 112
Dysentery, ... 113

Sub-Group (b).—Diseases dependent on Soil, Climate, etc.

Intermittent Fever, or Ague, ... 116
Remittent Fever, ... 121

Sub-Group (c).—Septic Diseases.

Erysipelas, ... 122
Septicæmia and Pyæmia, ... 127
Puerperal Fever, ... 129

Sub-Group (d).—Venereal Diseases.

PAGE
Syphilis, ... 131

Sub-Group (e).—Febrile Diseases communicable from Animals to Man.

Rabies—Hydrophobia, ... 136
Glanders and Farcy, ... 141
Splenic Fever, ... 142

GROUP *B*.—MISCELLANEOUS DISEASES.

Rheumatic Fever—Acute Rheumatism, . . 145
Gout, or Podagra, ... 151
Diabetes Mellitus, ... 157
Anæmia, ... 164
Rickets, ... 167
Purpura, ... 169
Scorbutus, or Scurvy, ... 170
Tuberculosis, ... 172
Hæmophilia, ... 175
Carcinoma, ... 176

SECTION III.

LOCAL DISEASES.

DISEASES OF THE RESPIRATORY ORGANS.

Preliminary Remarks, ... 179
Catarrh, ... 181
Hay Asthma, ... 183
Ozæna, ... 184
Laryngitis, ... 184

PAGE
Chronic Laryngitis, ... 187
Croup, ... 189
Varieties of Dyspnœa, ... 191
Tabular View of the Thoracic Regions, . . 193
Table of Morbid Thoracic Sounds, . . . 198
Diagnostic Table of Thoracic Diseases, . . . 201
Bronchitis, ... 202
Chronic Bronchitis, ... 207
Plastic, or Croupous Bronchitis, . . . 209
Asthma, ... 210
Congestion and Œdema of the Lung, . . . 212
Pleurisy, ... 213
Chronic Pleurisy, ... 219
Pneumonia, ... 221
Emphysema, ... 230
Pneumothorax, ... 233
Phthisis, ... 233
Cancer of the Lung, ... 241
Hæmorrhages of the Lung, ... 242
Table of Characteristic Sputa, ... 243

INFLAMMATORY AFFECTIONS OF THE HEART.

Pericarditis, ... 244
Endocarditis, ... 249
Myocarditis, ... 251

STRUCTURAL DISEASES OF THE HEART.

Anatomical Relations of the Heart, . . . 253
Valvular Diseases of the Heart, . . . 254
Table of Valvular Diseases, ... 262

PAGE
Hypertrophy of the Heart, ... 266
Atrophy of the Heart, ... 268
Fatty Degeneration of the Heart, . . . 269
Cyanosis, ... 272

FUNCTIONAL DISORDERS OF THE HEART.

Table of Functional Disorders of the Heart, . . 274
Angina Pectoris, ... 275
Exophthalmic Goître, ... 276
Thoracic Aneurism, ... 277

DISEASES AFFECTING THE DIGESTIVE SYSTEM.

Inflammation of the Mouth, ... 279
Tonsilitis, ... 281
Diseases of the Œsophagus, ... 282
Dyspepsia, ... 285
Gastritis, ... 288
Chronic Gastritis, ... 290
Gastric Ulcer, ... 291
Cancer of the Stomach, ... 293
Dilatation of the Stomach, ... 295
Enteritis, ... 296
Typhilitis—Inflammation of the Cæcum, . . 298
Diarrhœa, ... 299
Colic, ... 300
Lead Colic, ... 301
Constipation, ... 302
Obstruction of the Bowels, ... 303
Intestinal Worms, ... 305
Trichinosis, ... 308

DISEASES OF THE ABDOMEN.

PAGE

Peritonitis, ... 309

Chronic Peritonitis, ... 311

Ascites, ... 312

DISEASES OF THE LIVER.

Functional Disorder of the Liver, . . . 314

Acute Hepatitis, ... 315

Abscess of the Liver, ... 317

Cirrhosis of the Liver, ... 320

Fatty Liver, ... 324

Albuminoid, Waxy, or Lardaceous Liver, . . 325

Cancer of the Liver, ... 326

Acute Atrophy of the Liver, ... 328

Hydatid Tumours of the Liver, ... 330

Differential Diagnosis of Liver Diseases, . . 333

Jaundice, ... 334

Tabular View of the Causes of Jaundice, . . 334

Inflammation of the Gall Bladder, . . . 337

Gall Stones, ... 338

DISEASES OF THE PANCREAS.

Diseases of the Pancreas, ... 339

DISEASES OF THE SPLEEN.

Lardaceous or Waxy Spleen, ... 340

Leucocythæmia, ... 341

Hodgkin's Disease, or Lympho-Sarcoma, Malignant Lymphoma, Lymphadenoma, . . . 343

DISEASES OF THE NERVOUS SYSTEM.

PAGE
Inflammation of the Brain, ... 343
Chronic Inflammation of the Brain, . . . 349
Tubercular Meningitis—Acute Hydrocephalus, . . 350
Chronic Hydrocephalus, ... 357
Apoplexy, ... 360
Aphasia, ... 368
Sunstroke, ... 370
Delirium Tremens, ... 372
Insanity, ... 373
    (*a*) Idiocy, ... 373
    (*b*) Dementia, ... 374
    (*c*) Monomania, ... 375
    (*d*) Melancholia, ... 375
    (*e*) Acute Mania, ... 375
    (*f*) Puerperal Mania, ... 376
General Paralysis of the Insane, . . . 378
Epilepsy, ... 381
Laryngismus Stridulus, ... 385
Chorea, ... 387
Neuralgia, ... 389
Migraine, ... 390
Locomotor Ataxy, ... 392
Disseminated Cerebro-Spinal Sclerosis, . . 396
Glosso-Labio-Laryngeal Paralysis, . . . 399
Ménière's Disease—Auditory Vertigo, . . . 401
Progressive Muscular Atrophy—Wasting Palsy, . 402
Pseudo-Hypertrophic Paralysis, . . . 403
Infantile Spinal Paralysis—Acute Anterior Poliomyelitis, 404

PAGE
Scrivener's Palsy, ... 405
Mercurial Tremor, ... 406
Spastic Paralysis, Lateral Spinal Sclerosis—"Erb's" Palsy, 406
Diagnostic Table of Nervous Diseases, . . . 408
Hysteria, ... 410
Catalepsy, ... 411
Tetanus, ... 411
Diagnosis of Disease or Poison, . . . 414
Paralysis, ... 416
Hemiplegia, ... 417
Results of Injury to Brain, ... 420
Paraplegia, ... 421
Diagnosis of Hemiplegia, etc., ... 425
Bell's Palsy, ... 426
Table of Cranial Nerves, ... 427
Paralysis Agitans—Shaking Palsy, . . . 428
Diagnostic Table of Nervous Diseases, . . . 430

DISEASES OF THE SPINAL CORD.

Acute Spinal Meningitis, ... 435
Myelitis, ... 435
Acute Ascending Paralysis—"Landry's" Paralysis, . 436

DISEASES AFFECTING THE URINARY ORGANS.

Nephritis, ... 438
Bright's Disease—Albuminuria, . . . 441
Acute Bright's Disease, ... 442
Chronic Bright's Disease, ... 449
Table of Forms of Chronic Bright's Disease, . . 461

PAGE
Table of Kinds of Kidney, ... 462
Uræmia, ... 463
Diagnosis of Uræmic Coma, ... 465
Urine in Health and in Disease, . . . 465
Constituents of Urine, ... 467
Diabetes Insipidus, ... 469
Hæmatinuria, ... 470
Hæmaturia, ... 471
Chyluria, ... 472
Renal Colic, ... 473
Cancer of the Kidney, ... 474
Movable Kidney, ... 474
Hydronephrosis, ... 475
Diagnosis of Dropsy, ... 476
Diagnosis of Gravel, ... 477

DISEASES OF THE SUPRA-RENAL CAPSULES.

Addison's Disease, ... 480

DISEASES OF THE SKIN.

Papulæ, ... 482
Vesiculæ, ... 483
Parasitici, ... 487
Bullæa, ... 490
Pustulæ, ... 491
Squamæ, ... 492
Exanthemata, ... 493
Tuberculæ, ... 494
Diagnostic Tables of Skin Diseases, . . 486, 488, 489

DIETS FOR VARIOUS DISEASES.

PAGE
Diabetes (Pavy), ... 496
Fever and Inflammatory Diseases, . . . 496
Gastric Ulcer, ... 496
Gout, ... 496

PRESCRIPTIONS.

Anthelmintics and Cathartics, . . . 497
Antispasmodics, ... 497
Astringents, ... 498
Diaphoretics and Diuretics, ... 498
Emetics and Expectorants, ... 499
Gargles and Inhalations, ... 500
Liniments, Lotions, etc., ... 500
Narcotics and Sedatives, ... 501
Refrigerants and Salines, ... 501
Stimulants, ... 502
Tonics, ... 502

Index, ... 503

THE STUDENT'S HAND-BOOK

OF THE

PRACTICE OF MEDICINE.

TO place in the hands of the student a collection of concise and reliable notes on the Practice of Medicine is the object of the writer. It will be necessary, among other things, to treat first of certain morbid states and processes as such, irrespective of the part or parts affected. This is rendered necessary by the fact that these morbid processes form the constituents which in various associations are the elements of all the diseases which will be dealt with in detail in subsequent pages of this book.

The classification will then be as follows:—

SECTION I.—*Morbid States and Processes as such, irrespective of the part or parts affected.*

SECTION II.—*Diseases of the whole body, and Diseases which may be distributed in several parts of the body at one time.*

This class of diseases will have to be further divided into groups and sub-groups, as follows. In former editions of this book the term General Diseases was given to this class:—

A. Diseases dependent on Morbid Poisons—

- (*a*) Specific Fevers. Diseases in this sub-group present certain peculiarities.
    1. They are accompanied with pyrexia, frequently with eruptions on the skin, and have a tendency to run a definite course.
    2. They are often contagious and infectious.
    3. One attack generally confers complete immunity from a second.
    4. They may occur epidemically.
- (*b*) Diseases in this sub-group are, as a rule, endemic, and depend largely on the configuration, soil, climate, etc., of the countries where they prevail.
- (*c*) Septic Diseases.
- (*d*) Venereal Diseases.
- (*e*) Febrile Diseases communicable from animals to man.

B. Not classified, or Miscellaneous Diseases.

Section III.—Local Diseases—*that is Diseases attacking certain organs or parts of the body.*

SECTION I.

MORBID STATES OR PROCESSES AS SUCH, IRRESPECTIVE OF THE PART OR PARTS AFFECTED.

HYPERTROPHY.

Lat. *Hypertrophia.* Fr. *Hypertrophie.* Ger. *Hypertrophie.*

An increase in size, uniformity in the character of the normal constituents of an organ or tissue resulting from various causes, but chiefly from excessive nutrition, is known as hypertrophy. The form, structure, and functions of the organ are at the same time preserved. This condition is seen in hypertrophy of the heart subsequent to valvular disease; in the kidney when its fellow has become diseased. When the increase in the size of an organ takes place as a result of an overgrowth of one of its constituent elements loss of function, as a rule, occurs, and this increase in size is described as *false* or *pseudo*-hypertrophy, and is well seen in the disease known as pseudo-hypertrophic paralysis. Hypertrophy may be the result of an increase in the size of the individual elements, or in the number of the elements of an organ; in the former case it is said to be *simple*, in the latter *numerical.* To this latter form the term *hyperplasia* has been given. Mere increase in the size of an organ, it must be borne in mind, does not constitute genuine hypertrophy. A waxy liver, although increased in size, is really the subject of atrophy, for its normal tissue elements are decreased. The following are the conditions under which hypertrophy may occur. It may be compensatory or vicarious, as in the case of the loss of one kidney as just mentioned; increased supply of food, as in the case of hypertrophy of the spleen from frequent hyperæmia or congestion; diminished waste; and, lastly, to overwork or excessive stimulation, as seen in the muscles, or in the hardened cuticle, or corns on the hands of workmen. The causes of hypertrophy may be thus summed up : excessive nutritive supply, either as regards the quantity or quality of the supply; excessive stimulation or overwork, seen in the voluntary muscles—the overwork must not, however, be excessive; irritation applied to growing parts, probably by increasing the vascular supply, as seen in the lengthening of a long bone, of which one epiphysis remains ununited. Hypertrophy is also caused by intermittent pressure, seen in the growth of the cuticle, in corns on the feet and hands.

ATROPHY.

Lat. *Atrophia.* Fr. *Atrophie.* Ger. *Atrophie.*

The term atrophy has been applied to a condition, the result of defective or deranged nutrition, and in which the bulk of an organ or part is diminished in consequence of a diminution in the size *(simple atrophy)* or number *(numerical atrophy)* of the normal constituents of a part, unattended with marked degenerative changes of other kinds. The varieties just mentioned are often associated, the latter being an advanced stage of the former. Waxy liver and emphysema of the lung are both examples of true atrophy; for although both the organs are increased in size, yet the normal tissue elements are decreased in size and number. Atrophy may be either local or general; in the former the subcutaneous adipose tissue first disappears, then that in deeper parts. An amount of fatty *degeneration* is, as a rule, associated with atrophy. Nutrition defective both in quantity and quality, deficient vascular supply, pressure and disuse or cessation of the normal functions of a part, are the causes at work in the production of atrophy either general or local. Defective nutrition as a result of defect in the quantity of nutriment taken is seen in cases of starvation, the result of disease or accident; in children, from the administration of improper food. Atrophy from deficient vascular supply may be seen in the testis, when from any cause its blood supply is diminished; in bones the result of the pressure exerted by aneurisms or other tumours; and even in some organs, as in the kidney for instance, by pressure exerted by some abnormal growth of one or other of its normal constituents. The effect of disuse is seen in the stiffened, outstretched arm of certain Indian devotees, or in ordinary paralytic affections of the limbs. *Passive atrophy* is described as due to disuse or cessation of the functions of an organ; thus muscles in the neighbourhood of a stiff-joint atrophy, and so does the female breast after the cessation of the catamenia. Atrophy of muscles as a result of impaired nerve supply must not be here omitted, as seen in infantile paralysis, glosso-labio-laryngeal paralysis, etc.

HYPERÆMIA.

Lat. *Hyperæmia.* Fr. *Hyperémie.* Ger. *Hyperaemie.*

When there is an excess of blood in an organ or part, accompanied with more or less dilatation of the vessels, hyperæmia or congestion is said to be present. It may be either *active* or arterial, *passive* or venous, the latter due to mechanical obstruction, to the outflow of the blood through the veins, from disease of the heart or lungs. Active hyperæmia is the result of vaso-motor paralysis, which may be either directly or reflexly produced. As results of active hyperæmia we have increase of temperature in the part, increase in the size of the arteries, with thickening of their walls and in the case of the brain, great excitability, etc. There is also increase in the function of hyperæmic glands, except in those whose functions are only increased by nervous stimulation, the submaxillary glands being taken as a case in point. In the case of venous congestion the tissue elements may atrophy where the increased pressure in the capillaries can take effect on the surrounding cells, as seen in cyanotic atrophy or *nutmeg liver.* The walls of the veins are thickened, and the liver cells destroyed. Pigmentation may also occur when blood has escaped from the vessels, as seen in the centre zone of the liver in the above mentioned form of cyanotic atrophy of the liver. In hyperæmia the vessels of the part are also affected, there is dilatation with decreased velocity of the blood current, as in mitral stenosis. True fibrous thickening of the walls with deposit of pigment, as seen in "brown induration" of the lung. And lastly, hæmorrhage may, though rarely, occur from venous congestion, except in the brain and lungs where the support to the vessel walls is least. Where the valves give way in the case of varicose veins, and ulceration near the vein weakening its walls, hæmorrhage may also occur.

The term "cyanosis" has been applied to all cases where the hyperæmia is due to venous congestion, and in reality means "blueness" of the part, as in cyanotic cancer, cyanotic atrophy of the liver, etc. Cyanosis due to malformation of the heart is treated elsewhere.

In close connection with this subject come the two conditions known as thrombosis and embolism.

Thrombosis is due to the formation of a "thrombus" or clot in the vessels during life, especially in the veins where the blood pressure is lowest. Thrombosis is the presence of a clot or thrombus at the point of formation; embolism, is the formation of a clot or embolus at one point of the circulatory apparatus, but carried by the blood currents to another part. The conditions affecting the vessels most favourable to thrombosis are obstruction to the blood current by external pressure, the action of gravity as in varicose veins, and cerebral sinuses, narrowing of the vessel as in auriculo-ventricular stenosis, followed with coagulation in the auricle, loss of elasticity in the walls of a vessel, roughening of the walls by "vegetations," either atheromatous or syphilitic; and lastly, inflammation of the veins in the vicinity of abscesses, phlebitis, etc. With regard to the blood we may have an excess of fibrin-forming elements; *hyperinosis*, as in typhoid and other fevers; septic, in pyæmia, etc. The most common seats of thrombosis are the left auricle in mitral stenosis, polipi of the heart among the columnæ carnæ of the left ventricle. These may be a *post-mortem* formation. In the arteries, and then chiefly in the smaller ones, when their walls are diseased, or when an artery is in close connection with inflamed parts; in the small veins of the brain in fevers, in the pelvic and femoral plexuses in typhoid, etc.

The characters of a clot formed *during life*, if large and quickly produced, are a dark reddish soft mass, smooth and glistening, removable from the walls of the vessel, presenting here and there little pale points. If formed *more slowly*, the clot is of a dirty white colour, firm in consistence, and distinctly laminated and fibrinous, which under the microscope shows a fibrinous layer or network containing chiefly altered white blood corpuscles. When formed *after death*, the clot is loose, soft, and easily broken down, not laminated but composed of two layers, the one red, the other yellowish white, and which, under the microscope, presents a meshwork of fibrin containing both red and white corpuscles. These characteristics will help to diagnose *ante-mortem* from *post-mortem clots*, and should, therefore, be borne in mind during all inspections for medico-legal purposes. The results of thrombosis as regards the vessel itself and the clot must now be briefly considered.

In the *vessel* the chief changes take place in the inner coat, the middle suffers from fatty degeneration and becomes absorbed, and the outer coat thickened from increase of the connective tissue. As a result of these changes the elasticity of the vessel is impaired, there is a tendency to rupture and the formation of aneurism. The walls of the vessel are thickened when the clot becomes "organised;" but should the clot be septic, ulceration may ensue. With regard to the *clot*, it may remain unchanged at the point of formation, or undergo fatty degeneration either centrally or as a whole. If septic it breaks down into a pulpy straw-coloured mass, portions of which may be carried into different parts of the body. As a rule, however, the clot becomes "organised;" the chief changes, however, begin, and for the most part are found, in the walls of the vessel. The clot first becomes adherent to the wall of the vessel, and then the process of organisation begins, and in six or seven days vessels pass from the vasa vasorum of the middle coat into the clot and there form a network. The liquid portions of the clot are absorbed, and the solid undergo fatty degeneration; and lastly, the fibrous tissue contracts on the newly formed blood vessels, and the portion of the artery in which the clot was formed is reduced to a fibrous cord.

Embolism, as just said, is due to a clot or foreign body carried from one part of the circulatory system to another, and, as a rule, occurs in the smaller arteries and the capillaries, and sometimes in the portal vein. The changes in the clot and vessel are similar to those in thrombosis. The results are obvious—increased colateral circulation where such is possible; where not possible, degeneration and death of the part normally supplied by the plugged artery. Mr Symington, of Edinburgh, has lately shown that beyond the anastomosis which takes place in the "circle of Willis" the arteries of the brain do not anastomose, hence degeneration of the portion of the brain supplied by the middle cerebral artery when that artery is plugged with a clot, the result being aphasia, etc. etc.

HÆMORRHAGE.

Hæmorrhage may occur in several ways. Thus we may have hæmorrhage from the nose, *epistaxis* (επι upon, and σταζω I flow drop by drop); *hæmaturia*, blood from the urinary passages (αιμα blood, and ουρεω make urine); *hæmoptysis*, spitting of blood (αιμα blood, and πτυω I spit); *hæmatemesis*, vomiting of blood (αιμα and εμεω I vomit); *melæna*, blood from the bowels (μελας black, and αιμα); *metrorrhagia*, blood from the uterus (μητρα the womb, and ρηγνυμι I break forth); menorrhagia, synonymous with the last (μην a mouth, and ρηγνυμι I flow fiercely). Under any circumstances hæmorrhage results from one of two causes, either the direct rupture of a vessel, or the gradual oozing out of the solid and liquid portions of the blood through the walls of the vessels from causes not yet well ascertained. The latter is known as *diapedesis* (διαπηδαω I leap through).

The two forms of hæmorrhage, however, which more particularly belong to the province of the physician are hæmoptysis and hæmatemesis; and as it is of importance to clearly distinguish the one from the other, we shall give their points of difference in a tabular form.

HÆMOPTYSIS.	HÆMATEMESIS.
The blood, which is of a bright scarlet, comes from the lungs. It is brought up by coughing, is fluid and frothy, from admixture with air. It has an alkaline reaction. Hæmoptysis frequently occurs during the first and last stages of phthisis, and is not of unfrequent occurrence in valvular disease of the left side of the heart, and sometimes when there is congestion of the liver with an absence of lung disease. This should be borne in mind.	In this case the blood comes from the stomach, and is expelled by vomiting. It is of a dark purple or black colour, more or less coagulated and mixed with the food. It has an acid reaction. It may be due to congestion of the liver, cirrhosis, and consequent impediment to the portal circulation. It may also occur as a vicarious form of menstruation, or from an ulcer or cancer of the stomach, in which latter case

It accompanies, and is followed by, *pulmonary apoplexy*—a bad term, but which is used to imply a state of extravasation of blood into the air-cells and tissue of the lung, which produces crepitation in the part till the blood is absorbed.

The treatment of hæmoptysis will consist in impressing on the patient the necessity for perfect rest in a semi-recumbent posture, and he must be forbidden to speak. The room should be kept cool. Ice may be kept in the mouth, or a bladder containing ice applied to the chest. Cold drinks should alone be allowed, and medicines, as for instance the acetate of lead, ergot, F. 43, gallic or tannic acids, given at frequent intervals.

it is followed by melæna, that is, the passage of a portion of the blood with the stools. The blood becomes changed, is very black, and so altered as to look like tar, or even soot.

In the treatment, ice should also be allowed. If there be much congestion of the liver, a good saline purge, to relieve the portal circulation, will be necessary. Styptic medicines—alum, turpentine, and others—may be given, if the hæmorrhage still continues severe.

DROPSY.

Lat. *Hydrops*. Fr. *Hydropisie*. Ger. *Wassersucht*.

Definition.—A collection of serous watery fluid in serous cavities of the body, or in the areolar tissue, without the presence of inflammation, and due to excessive transudation from altered conditions of the blood, altered pressure relations, disease or obstruction of the lymphatics.

Various names have been applied to denote the position of the effusion in dropsy; thus, when in the peritoneal cavity, it is known as *ascites;* in the pericardium, as *hydro-pericardium;* in the pleura, *hydro-thorax; hydro-cephalus*, dropsy of the brain; when the areolar tissue becomes infiltrated, and the infiltration partial, *œdema; anasarca*, if it be more general.

Dropsy, as held by some, is not a disease in itself, but rather a symptom of disease in one or other of the internal organs. The general composition of dropsical fluids is as follows—Water, serum albumen, serum globulin and fibrinogen, fats, urea, and organic salts. The dropsical fluids which contain the most albuminous matters are those of the peritoneum and pleural cavities. As a rule, general dropsy depends upon hydræmia, which is produced when the elimination of water by the kidneys is lessened. The blood then becomes more watery, and the excess of the watery portion exudes through the vessels, with dropsy as a result. As the walls of the vessels are pervious, it follows that alteration of pressure is an important factor in the production of dropsy. Thus increased arterial pressure diminishes transudation, but increased venous pressure promotes it; the like result also follows on increased capillary pressure from vaso-motor paralysis.

Dropsy may then be due to:—

1. Disease of the heart. Cardiac dropsy, the result of diminished arterial and increased venous pressure, shows itself first in the feet and hands, the former "pitting" on pressure. From these parts it spreads over the body.

2. Disease of the liver. Hepatic dropsy, due to increased venous pressure, is known as ascites or peritoneal dropsy.

3. Congestion of the kidneys, giving rise to acute renal dropsy; first seen in the face, especially in the lower eye-lids.

4. Disorganisation of the kidneys, giving rise to chronic renal dropsy. In this case the dropsy is believed to be due to the transudation of fluid through the thickened walls of the capillary arteries, assisted by the increased blood pressure and stagnation, the result of the general capillary fibrosis.

Hepatic and cardiac dropsy are both the result of a mechanical impediment to the circulation of venous blood, leading to the exudation of a watery fluid through the capillaries. If a large venous trunk be subjected to pressure, œdema of the part below the

impediment is the result. Let the student suppose a pervious earthenware pipe through which a fluid is rapidly flowing. As long as a certain velocity is maintained there is no leakage, but let the current be arrested and transudation of the contained fluid will commence. Again, increase the velocity, the transudation will cease, and the previous transuded fluid will be drawn into the current. The effect of digitalis in passive dropsy, due to a weak or irregular acting heart, is thus explained; it slows and strengthens the heart, increases the blood pressure, and thus promotes the rapid circulation of that fluid. The diuretic action of the drug is due to the above mentioned action.
and is then due to the feebleness of the circulation

Lastly, dropsy may occur without organic disease, and thinness of the blood, which, according to Ringer, is due to a want of albumen in the blood, which reduces the affinity of the blood for the fluids in the parenchyma, and hence these accumulate and produce dropsy. This condition occurs most frequently in chlorotic girls and elderly females. The dropsical effusion is generally in the form of an anasarca. The treatment of this form of dropsy will consist in the administration of one or other of the preparations of iron and stimulating diuretics. The general treatment will be considered when the diseases which give rise to dropsy come under notice in their order.

INFLAMMATION.

Lat. *Inflammatio.* Fr. *Inflammation.* Ger. *Entzündung.*

DEFINITION.—The condition of a part or organ of the body brought about by local or other causes and characterised by pain, heat, redness, swelling, and some impairment of function in the part attacked.

The four cardinal characteristics of inflammation adopted by ancient medical writers were thus expressed—*dolor*, *robor*, *tumor*, *cum calore;* to which has now been added a fifth, *impairment of function in the part attacked.* These have been further classified into the *objective* symptoms, redness, swelling, and heat; *subjective* pain or tenderness and impairment of function. Dr Burdon-Sanderson has defined inflammation as "the succession of changes which occurs in a living tissue when it is injured, provided that the injury is not of such a degree as at once to destroy its structure and vitality." We shall now briefly consider these changes. The first of these in point of time may be described as due to vascular disturbance in the part. The arteries are first seen to dilate, in some cases, to nearly twice their size; later on, a like dilatation takes place in the veins. The blood current at the same time becomes quickened, but this temporary acceleration is soon arrested, the leucocytes falling out of the main stream adhere to the sides of the vessels till at last the vessels become choked by both red and white blood cells, and stagnation or *stasis* is the result. But while this process to stagnation is more or less rapidly going on, small rounded bodies in connection with leucocytes within the vessels are seen on their outer surfaces. These in process of time make their way through the coats of the vessel, become free, but still lie close to the outside of the vessel. This process of *emigration* must partly be referred to the power of spontaneous locomotion possessed by the leucocytes themselves, and partly also to their being under pressure from the blood. It has also been suggested that the migration of the leucocytes is due to the colloidal nature of their structure in common with that of the vessels, and that this migration is assisted by their amœboid action. Following close upon the above, and in some cases probably contemporaneously with it, we have the stage of exudation, in which fluid is poured out, and this together with the corpuscular elements just mentioned constitute the so-called *inflammatory exudation.* As it has been shown that the leucocytes can appropriate portions of colouring matters, Dr Hilton Fagge was of opinion that the migration of the leucocytes serves the special purpose of "clearing away such portions of an injured structure as are no longer fit to remain," a view more than suggested by the late Professor Goodsir in 1845. The tissue changes in an inflamed part are very complex, but they may be divided into two classes, that of *impaired nutrition* in the part itself, and of *increased nutritive activity* in the parts around. Inflammation may terminate by *resolution*, in which case the products of exudation are absorbed and the part regains its normal condition; by *suppuration*, in which pus is formed, and an abscess may be the result; by *ulceration*, in which the superficial tissues are destroyed; and, lastly, *granulation* may result, in which the damage previously effected by the inflammatory processes is repaired by the formation of a cicatrix of fibrous or other tissue.

Of the symptoms of inflammation before mentioned the pain is probably due in the first place to pressure on the nerves by the distended vessels; and, secondly, to the exudation. The redness to increased vascular supply, and also to some effusion of blood from ruptured blood vessels. Swelling is the result of vascular turgescence and of the exudation. The cause of the increased heat of an inflamed part is probably due to the increased vascularity of the part, the rapidity of the blood current, and to abnormal oxidation of one or more of the tissues of the part. The last of these causes has not, however, been verified by experiment, and it is probable that the heat of an inflamed part is never higher than the internal temperature of the body. The inflamed part is *only* locally hotter *than* the surrounding parts due to the first two causes just mentioned. Inflammation may be either *acute* or *chronic*, *difuse* or *circumscribed*, *simple* or *infective*. It may also be *suppurative*, *ulcerative*, *gangrenous*, or *adhesive*. In mucous membranes we may have *catarrhal*, *croupous*, and *diphtheritic*. The character of the exudation depends on the relative proportion of the fluid and solid constituents. The exudation consists of liquor sanguinis and cellular elements, and is said to be *serous*, when thin and watery; *fibrinous* or "plastic," when the fibrin elements are large; *purulent*, when turbid and contains pus cells; *hæmorrhagic*, when blood is present. When the special tissue of an organ is attacked the inflammation is said to be *parenchymatous;* when the network, *interstitial;* and *catarrhal*, when a mucous membrane is involved.

DEGENERATION.

Lat. *Degeneratio.* Fr. *Dégénérescence.* Ger. *Degeneration.*

Definition.—Alteration in the *quality* of a tissue either due to chemical or molecular changes of its protoplasm, so that by such alteration there is a tendency to ultimate destruction or death of the tissue, preceding impairment of function.

Degeneration differs from atrophy in this, that nutrition in the former is altered in *quality;* in the latter, in *quantity*. As a result we have in atrophy waste of tissue in excess of the assimilation of new material; in degeneration a metamorphosis of the tissue and an abnormal substance exists which is not consumed. Thus the vitality and functions of the tissue are impaired. The terms "parinchymatous" or "granular degeneration," "albuminous infiltration," "molecular degeneration," or lastly "cloudy swelling," by Virchow are applied to the first changes in commencing degeneration. The change thus mentioned occurs most frequently during the course of the specific fevers, typhoid, typhus, and acute rheumatism, also in pyæmia, erysipelas, etc. And in these diseases the organs most liable to attack are the heart, kidneys, liver and lungs, and even the muscles. The causes at work in the production of "cloudy swelling" are high temperature, as Dr Wickham Legg has produced it in animals exposed to high temperatures, early inflammation, and deranged nutrition. To the naked eye, as in the liver where the change is generally most marked, the organ appears slightly enlarged and anæmic, and of a dull opaque greyish-white colour. Examined under the microscope the cells are swollen and granular, and in some cases contain fatty particles. In some cases the organs attacked recover their normal appearance, in others fatty degeneration supervenes.

In order to distinguish "cloudy swelling" from other degenerations, the following table may be of use :—

1. Cleared up by acetic acid.	These show that the granules are not fatty in nature.
2. Not affected by ether or chloroform.	
3. Not stained black by perosmic acid.	
4. The granules are not stained by carmine or logwood.	This shows that the granules are not composed of protoplasm.—*Catechism of Pathology.*

Degenerations may be divided into two classes. The *Metamorphoses*, including mucoid, fatty, colloid, lardaceous (?). The *Infiltrations*, fatty, calcarous, and pigmentary.

FATTY DEGENERATION.

Lat. *Adiposa.* Fr. *Graisseuse.* Ger. *Fettige.*

Definition.—A process by which there is a gradual replacement of the protoplasmic elements of a part by molecular fat.

Due to certain causes to be presently mentioned, the normal protoplasmic constituent of the tissues is so changed that its molecules are converted into fat. It is, in fact, a destruction of the normal histological elements, and thus differs from "fatty infiltration" in which the cells take up into their substance particles of fat which do not form their normal constituent. In the latter case, the cells may again become healthy by parting with the accumulation of fat in them ; in degeneration this cannot take place, as it is the cells themselves that have undergone change.

Fatty degeneration may be either *physiological*, as is the case in the uterus after delivery, the secretion of milk, etc., or *pathological*, as in the heart and other organs. The results of fatty degeneration are softening and brittleness of the part, decrease in its specific gravity, atrophy and a change of its colour to a greyish yellow or brownish yellow tint. Under the microscope the cells of the affected part may be seen swollen, but, as a rule, wasted and full of oily granules.

Fatty degeneration occurs most frequently in highly organised tissues, muscles, and in parts where the nutrition has been arrested. It may also be present where the obstruction to the blood supply has been rapid, as in tubercles, infarcts, etc., and also in cases where the growth of a part has exceeded the nutritive supply; this is seen in some tumours of rapid growth. This form of degeneration is never general, as the tissues always give indications of the presence of their normal constituents. The presence of fat may be detected by the granules being soluble in ether or chloroform, unaffected by acetic acid and blackened by osmic acid. The causes at work in the production of a pathological accumulation of fat in the tissues are as follow :—

1. Excess of fat in the food or of substances readily converted into fat.

2. When oxidation is so imperfect that the fat of a normal diet is imperfectly consumed.

3. When there is incomplete oxidation of the nitrogenous constituents of food.

4. When, during the process of nutrition, the fat liberated from the protoplasm cells is imperfectly oxidised and ultimately replaces them.

The conditions under which fatty degeneration occurs are :—

I. Defective nutrition.

1. *General*—

(*a*) Wasting or exhausting diseases, phthisis, anæmia, etc.

(*b*) Starvation from want of food or disease of the alimentary canal.
(*c*) Defective assimilation, as in old age.

2. *Local*—

Diminished blood supply from disease of vessels.

II. Loss of function, as in nerves and muscles.

III. Poisons—phosphorus, lead, antimony, arsenic, alcohol, etc.—*Catechism of Pathology.*

Caseation is closely allied to fatty degeneration, to which it not unfrequently forms a sequel. It appears to be due to a drying up of degenerated tissue, the result of deficient blood supply, and forms a cheesy mass composed of shrivelled cells, fat globules, and cholesterine. These "cheesy" masses are found chiefly in the lungs as a result of tubercles, but their presence may also be detected in syphilitic gummata, dried up abscesses, etc. They may also become the seat of calcareous deposits occurring in the *middle* or muscular coat of the arteries of old people, and must not be mistaken for atheroma which is a disease of the *inner* coat.

MUCOID DEGENERATION.

Lat. *Mucosa.* Fr. *Muqueuse.* Ger. *Schleim.*

Definition.—The transformation of the albuminoid *constituents* of the tissues into mucin, a reversal of the fœtal process in which the higher is developed from the lower.

As the development of the body proceeds, the primary tissues, except in the case of the *vitreous humour* which is permanently a mucoid tissue, become developed into higher forms of connective tissue. In mucoid degeneration the degenerative process is closely allied to those processes normally occurring in the fœtus, only reversed; the tissues becoming converted into a soft mucilaginous jelly-like substance giving the reactions of mucin. It differs from albumen in not containing sulphur, is insoluble in excess of acetic acid, and is not precipitated by boiling, by corrosive sublimate, or by tannin. As a physiological process it occurs in the secretion of mucous in catarrhal affections of mucous membranes.

Mucoid degeneration may occur in cartilage, in connective tissue, either formed or in the process of forming, and in myxomatous tumours.

COLLOID DEGENERATION.

Lat. *Colloides.* Fr. *Colloïde.* Ger. *Colloid.*

Definition.—A form of degeneration in which the abuminoid *contents* of the cells are converted into a peculiar "colloid" materia differing from "mucin" in containing sulphur and not being coagulated by acetic acid or alcohol.

The colloid material is colourless, translucent, has the consistence of half-set glue, and first makes its appearance as small lumps *within* the cells, and which it ultimately fills. The cells in time coalesce to form larger masses of jelly-like material, readily visible to the naked eye. The cells are destroyed, the intercellular tissues atrophies, and cavities containing the colloidal substance are formed. Colloid change is most commonly met with in the thyroid gland and in the lymphatic glands, it has also been found in colloid cancer of the stomach.

LARDACEOUS DEGENERATION.

Lat. *Lardacea.* Fr. *Lardacée.* Ger. *Speckige oder amyloide.*

Definition.—A degenerative tissue change, probably the result of their altered nutritive properties, by which proteid substances circulating in the blood are combined with the altered tissues to form the peculiar lardaceous or waxy material.

Lardaceous, albuminoid, or amyloid degeneration is one of the most important of the tissue changes. It may be found in nearly every tissue and organ, but the organs most frequently attacked are the liver, spleen, and kidneys. The conditions under which it most frequently occurs are in cases of prolonged suppuration, chronic phthisis, and constitutional syphilis. The parts affected are swollen, translucent, with a peculiar homongenous glossy or waxy appearance, and with a dilute solution of iodine strikes the characteristic mahogany brown colour, turned to violet or a slate blue colour on the addition of very dilute (2 per cent.) solution of sulphuric acid. The healthy parts of the organ are stained purplish blue, the waxy parts pinkish or ruby coloured by methylaniline violet. The amyloid material differs from the starches in containing nitrogen, and is not, as suggested by Dickinson, de-alkalised fibrin, its chemical constitution being against that theory. Amyloid degeneration, to whatever causes it may be due, attacks primary the arterioles and capillaries, first the middle and subsequently the inner coat, and then the other tissues of the organ.

CALCAREOUS INFILTRATION.

Lat. *Calcarea.* Fr. *Calcaire.* Ger. *Kalkföunige.*

Definition.—An infiltration of the tissues with calcareous materials.

Simple calcification, just mentioned as occurring as an ultimate change in fatty degeneration, must be distinguished from ossification. The latter is an *active* process in which there is an intimate union between the cellular elements of the tissue and the calcareous materials in which the latter cease to be visible; the former is essentially a *passive* process, being a pure infiltration of calcareous particles without any nutritive activity of the part. In some cases, as in the lung, calcification is a useful process, as the calcified part undergoes no further change. The calcareous particles, chiefly lime and magnesia salts, as phosphates and carbonates, occur both in the cells and intercellular substance of the part, and are detected by their opacity, dark black colour, and irregular outline, when viewed under the microscope by transmitted light, and their solubility in the dilute mineral acids.

PIGMENTARY INFILTRATION.

Lat. *Pigmentaria.* Fr. *Pigmentense.* Ger. *Pigment.*

Definition.—An infiltration of pigment in the tissues.

This form of degeneration consists in the infiltration of the tissues due to the deposit in them of colouring matters derived from the blood. The skin of the negro and choroid coat of the eye are instances of permanent pigmentation. Certain tumours, "melanotic sarcoma," often are characterised by this form of degeneration. The deposit of particles of carbon in the lungs of miners, etc., must not be mistaken for true pigmentary infiltration.

FATTY INFILTRATION.

Definition.—A condition in which the cells of a part become abnormally loaded with fat, but the tissues themselves remain for a time at least normal.

Fatty infiltration differs from fatty degeneration as before mentioned. As a physiological process it occurs in the growth of adipose tissue, and, as the fat is only stored up in the cells, they are not affected by its removal. When the infiltration becomes excessive, due to over-feeding and sedentary occupations, injury may result to the organs infiltrated.

SECTION II.

GROUP *A*.

DISEASES DEPENDENT ON MORBID POISONS.

SUB-GROUP (*a*).—*PYREXIÆ—FEVERS.*

Fever may be defined as an acute affection of the system of a nature more or less complex. It may occur *idiopathically*—that is, as a primary disorder—scarlatina, typhoid, etc.; or *symptomatically*—that is secondarily, in connection with other diseases as part of their phenomena—pneumonia, etc. Some, indeed, have contended that idiopathic fever does not exist, and that fever is always a symptom of some local lesion. This view is not now entertained by the best authorities. Vital and chemical action raised above the healthy standard, probably depending upon alterations in the nervous system, together with some disturbance of the processes by which heat is removed, results in fever, marked by an abnormal rise in temperature.

The normal temperature of man may be taken at 98·4°, but this is subject to two variations known as the "diurnal variations," a *minimum* between 6 and 7 o'clock A.M., and *maximum* about 6 P.M. These variations, apparently independent of food, closely follow the diurnal variations noticed by the meteorologist, but in man they appear to follow the meteorological by an hour or two. The presence of these variations should be borne in mind when taking the temperature for clinical purposes.

Due to vital and chemical processes, resulting in oxidation or combustion of nitrogenous and carbonaceous substances furnished to the blood by the tissues, but chiefly by the food, the natural heat of the body is maintained. The carbonic acid, urea, and uric acid products of this combustion are—the first, eliminated by the lungs; the two last, by the kidneys. The formation of carbonic acid is effected by the red blood cells, the urea and uric acid being probably formed from albumen in its passage through the liver, spleen, etc., and even through the cells of the blood itself. The fixed albumen of the muscles, nerves, and other tissues is transformed into urea, and probably, also, the so-called store albumen existing in the blood. The urea formed in the body is always increased in fever, even though less food be taken than in health, and its formation and elimination is always greatest when the temperature of the body is greatly increased. The temperature of fever differs, however, in its origin from that of health, in that the elimination of nitrogen is regulated by the food taken; whereas, in fever the amount eliminated is vastly in excess of that taken. The result of this is that the muscles waste and the brain atrophies, and an increase of cerebral fluid takes the place of the wasted brain. Waxy degeneration of the muscles is present in all fevers of a severe type. The glandular organs, liver, spleen, kidneys, etc., are the only portions of the body that do not waste, and these become enlarged and congested, owing to their increased functions. The white blood cells are also increased in number and in size. The elimination of carbonic acid is increased in fever, due to the increased frequency of the respirations, although the actual amount of carbonic acid in each portion of expired air is less than in health. This may account for the rapid disappearance of fat in fever. Fever is defined by Virchow as "that state of the body in which there is an increase of temperature above the normal;" but this elevation of temperature must be permanent, not merely a temporary condition. Simple elevation of temperature, with no concomitant phenomena, seldom or never occurs, and, therefore, according to the above definition, there is, strictly speaking, no such thing as a simple primary or idiopathic fever. The nearest approach to it is found in the affection known as febricula, to be presently described.

As to the causation of continued fevers opinions vary greatly. Some, with Murchison, maintain that it is possible to arrest the spread of continued fevers, and even to prevent their origin, and claim for these diseases a spontaneous generation. Others deny the origin *de novo*, and describe and figure certain vegetable parasites to which they attribute the origin of these diseases. Murchison distinctly says, "There are certain contagious diseases, such as erysipelas, pyæmia, and puerperal fever, whose origin *de novo* may be said to be a matter of almost daily observation, and which, in fact, we have almost the power of generating at will." He then proceeds to show that pyæmia may be generated from an unopened abscess to which atmospheric germs could not possibly have gained access, and he concludes "that there are good grounds for believing that contagious fevers have occasionally an independent origin." Liebermeister, on the other hand, says, "We have gradually reached the conclusion that it is only where the specific germ of the disease exists by itself, or has been introduced, that those anti-hygienic factors become active, and may then be capable of occasioning an enormous extension of the disease. The germ, however, is not produced by spontaneous generation. The spontaneous origin of small-pox, measles and scarlet fever could scarcely find a defender now. Perhaps the time is not far distant when the doctrine of the spontaneous origin of typhus fever, dysentery, typhoid fever, etc., will be universally refuted." He further remarks that, "from the specificness of infectious diseases we reach the natural conclusion that they never originate spontaneously, but are dependent upon a transmission, a continued propagation, of the disease poison."

The following, when there is no local disease adequate to account for them, may be taken as a tolerably accurate enumeration of the phenomena which usher in and attend an idiopathic, essential, or primary fever:—There is in most cases a period of incubation, which is more or less prolonged, and which varies in each particular fever. This state is succeeded by a feeling of chilliness, accompanied with rigors. The pulse becomes quickened, the respirations increased in frequency, and the temperature of the body greatly augmented. The blood becomes impoverished, due to the diminished quantity of its normal constituents. The digestive functions are deranged, the desire for food is entirely lost, but there is intense thirst. This latter symptom, although constant attempts are made by the patient to relieve it by large draughts of fluid, still continues, yet very little of the fluid taken is eliminated by the skin or kidneys. The urine becomes scanty and thick, due to a diminution in the quantity of water and to an increase in the urea and uric acid. The chlorides are, however, diminished, and in pneumonia the diminution begins with the stage of hepatisation, their gradual reappearance marking the stage of resolution. The excretion of the chlorides is, however, greatly increased during the cold and hot stages of ague. Although the urine, as a rule, contains more solids than in health, yet at times, in the course of some fevers, the solid constituents sink below the normal. To the above symptoms may be added a feeling of nausea, then vomiting, headache, lassitude, increasing emaciation in spite of the nourishment taken, pains in the limbs, restlessness, insomnolency, and often delirium. These symptoms are present in all the disorders to which the term fever is applied, and are combined with peculiar local lesions in the respective diseases, *e.g.*, with sore throat and a characteristic rash in scarlatina, with intestinal ulceration and a characteristic rash in typhoid fever, with inflammation of the parenchyma of the lung in pneumonia, and with affections of the joints and fasciæ in acute rheumatism.

Fever may terminate in three ways—by *crisis*, that is, by an abrupt and sudden termination to the symptoms, as is the case in some fevers where the intense heat of the skin gives place to a profuse sweat, and is that point in the course of fever when the morbid products in the body have arrived at a condition to be eliminated by one or more organs; by *lysis*, where the change is gradual and almost imperceptible; and lastly, by a combination of both.

Fever may cause death in three ways:—(1) by the blood becoming poisoned; (2) by emaciation, and subsequent exhaustion; (3) by the abnormal and persistent increase in the temperature of the body.

The doctrine most reconcilable with our present knowledge is, that fevers consist of a primary blood disorder, the nervous system becoming secondarily affected, together with increased oxidation, this probably taking place in the muscles. The initiatory rigor is perhaps the first evidence of nervous implication in the patient, but in the subsequent phenomena both blood and nervous system are alike implicated. The following may be taken as an epitome of the reasons for adopting this view:—

(*a*) The introduction of a blood poison is rendered probable by the existence of a period of incubation.

(*b*) Fevers appear to be governed by certain laws which limit their existence; this fact may point to certain processes in the blood, which in time become exhausted.

(*c*) Some of them may be produced by inoculation—that is, the introduction of a tangible virus. This is the case with small-pox. From this we infer that in other varieties of fever the same may occur.

(*d*) No known cause acting directly on the nervous system has as yet been proved capable of producing any of the fevers.

(*e*) It is more than probable that most nervous disorders have their origin in a vitiated state of the blood.

Diseases of the nervous system are uncertain in their development and duration.

From the above, and from the difficulty of conceiving how external causes which give rise to fevers can possibly affect the nervous system prior to the blood, it is reasonable to suppose that the nervous phenomena which often attend fevers are induced by the primary blood disorder. Some have supposed that the nervous system during health regulates the oxidation which takes place in the body, and thus keeps the temperature below 100° F., and for this purpose they have suggested the existence of a nervous centre, which becoming paralysed, fever results. This is the theory of Virchow. Naunyn and Quincke have placed this "fever centre" in the medulla oblongata. Rosenthal and Sanderson have shown that the experiments of Naunyn and Quincke are not conclusive, and that the "centre" has not yet been found. Traube suggests that the vessels near the surface of the body becoming contracted, and less blood circulating through them, less heat is lost, and fever is the result. This theory is negatived by the fact, that when this contraction is artificially produced, fever, or any great degree of heat, is not produced. Marey opposes Traube, and suggests that the superficial vessels are paralysed, and that more blood circulates through them. This may explain the hot skin, but it does not account for the high temperature of the internal parts, which, according to this theory, ought to be colder from the amount of blood at the surface. Liebermeister maintains that fever is due to a change in the "normal function of heat regulation," and "one might imagine the index of the regulating machinery to be shifted upwards, so that it is 'set,' not at 98·4°, but at 101°, 102°, 103°, or even a still higher point." This, although it suggests the presence of a heat regulating apparatus, does not in any way enlighten us as to the cause or causes which produce the shifting of the "index of the regulating machinery." The theory has one advantage in showing that fever is a definite modification of a physiological process, and that pyrexia may be really salutary working its own cure by the destruction of the agents which set it up, as in the case of relapsing fever, in which it is found that the pyrexia destroys the *spirilla* which are now held to be the cause of the specific fever. Sanderson suggests, without propounding a theory, that the increased heat of fever is the result of increased oxidation, and that this increased oxidation takes place in the muscles. If muscular exertion is continued under conditions that prevent loss of heat from the body, fever is the result, and thus pyrexia is produced by rapid tissue change. Any temperature above 101° F. may be held to betoken fever, and it has been shown that even in the so-called "cold stage" of ague the temperature of the body is increased.

GENERAL TREATMENT OF FEVERS.

In the treatment of fever there are certain general indications which are worthy of attention. At the onset of the disease, when the specific character of the fever is as yet uncertain, an emetic or a mild laxative may be given followed by a diaphoretic mixture, F. 2. The bowels are thus relieved, and the action of the skin promoted. The feverish symptoms accompanying a catarrhal attack are often thus relieved, and not unfrequently an apparently severe catarrh may be thus cut short. In the case of children suffering from a feverish attack, I have frequently found that a hot bath at night comforts the little sufferer, and promotes sleep, the child being quite well in the morning. In giving purgatives, the mildest should, in the first instance, be alone used; for should the attack turn out to be one of typhoid, much mischief may be done by too free purgation. In the course of an attack of fever much care will be required in the treatment of the complications as they arise. One of the most important of these is hyper-pyrexia, or a continued and abnormal rise in temperature. The gravity of this state will depend on the nature of the fever, its probable duration and character. Thus, a temperature persistently high in typhoid, a fever of some duration, is of far greater gravity than a like temperature in relapsing fever. It must also be borne in mind that in some fevers—scarlet fever and relapsing fever—a high temperature is characteristic of the disease. We have, therefore, to bear in mind the duration and character of the fever before we decide as to our prognosis of its probable result. To regulate the temperature ice may be applied to the head, or the body may be sponged with cold or tepid water, but by far the most important anti-pyretics are the "wet pack," cold or tepid, and the cold bath. In the former the bed is first protected by a water-proof sheet, and on this is placed a dry blanket; on this again a blanket, wrung out in either cold or hot water, is placed, and then the patient is carefully folded up in the two blankets. In these he may remain for half-an-hour, be then removed, carefully dried, and put back to bed. The cold bath is a favourite method of treatment with Liebermeister in typhoid. It consists in placing the patient in a bath at a temperature of about 80° F., and then rapidly lowering it by the addition of ice to 60° or 50°. In this the patient may be kept for from fifteen to thirty minutes, but he should be at once removed on the slightest appearance of collapse, and means taken to assist a healthy reaction. In cases of marked and persistent hyper-pyrexia, this treatment has undoubtedly saved life. Quinine, in one large dose, 15 to 30 gr., given at night, has been recommended in the treatment of the hyper-pyrexia of typhoid. In typhus its use is doubtful. It is of great importance in the treatment of fever that the services of an experienced fever nurse be obtained, and that the nursing of the patient be entrusted as little as possible to his friends or relations. In the course of fever so many anxious and critical periods occur, that it is never well to leave to relations the duty of watching the sufferer. In the next place, a large and airy room should be provided, and if the house be situated in a street, it is as well to select a back room away from all noise. Some fevers—small-pox, scarlet fever, etc.—may with advantage be treated in tents, or wooden huts, which may be pulled down when the epidemic has passed. All carpets and useless furniture should be removed, and, if possible, the room should contain two beds, each bed being used for two or three days at a time by the patient, or he may be changed from one bed to the other daily. Only those who have been ill for some weeks, or even days, can appreciate the comfort of being moved from a hot and uncomfortable bed into clean sheets. The nurse need never be required to sleep in the room, but during the hours allotted to her for sleep she should have a room to herself. The sick-room may be ventilated by a fire, and the temperature regulated by a thermometer.

Administration of Medicines, etc.—As it is noted that towards night fevers increase in severity, the pulse rising instead of falling, and the skin, ceasing to act, becoming hot and dry, so it is argued, and with justice, that during the afternoon and evening are the times when febrifuge medicines should be administered. The object in view is to increase the discharge of urea, and also to augment secondarily the action of the skin. The excretion of urea is abnormally large whilst the feverish symptoms last. The natural tendency is for the excretion of urea to become less towards evening; but, as before said, the excretion is increased during the fever, which is greatest towards night, and if the discharge of the urea be prevented, dangerous complications result. Our object, therefore, is to help the discharge of the urea and promote the action of the skin. Febrifuge medicines and diluents are therefore indicated. Purgatives do not meet the requirements of the case, as they remove carbon, but not urea. On the other hand, the time for administering food and alcohol is in the fore part of the day. But when the fever has entirely left, during the subsequent debility and convalescence, nourishment should be given in the evening and during the night. Stimulants and tonics are best given morning and evening, and even during the night should the patient awake. The patient may even with advantage be aroused from sleep to have nourishment administered.

The Diet in Fever.—The diet in fever is important. It must always be borne in mind that, in a case of fever, we are dealing with a disease that may last from three to four weeks and more. It therefore requires more than ordinary care to regulate the amount of nourishment given by those critical periods just when the recovery or death of the patient may occur. Some years ago it was the custom "to starve a fever," and there can be no doubt that many deaths resulted from this method of treatment, for many of the symptoms which were then held to indicate inflammation of the brain or its membranes, or of the stomach, were in reality the indications of protracted abstinence. Thus, pain in the head, epigastric tenderness, thirst, vomiting, determination of blood to the head, suffusion of the eyes, headache, sleeplessness, and delirium, are the symptoms of the deprivation of food. Fever patients have a great aversion to take food—it is, therefore, necessary to insist on its being taken as a medicine. In the early stages, if the patient be strong and robust, barley-water and milk, with occasionally some beef-tea or mutton broth, should be ordered, and there is no objection to gruel made with milk. In typhoid fever only slops should be given, and a return to solid food made with the *utmost* caution. As the disease progresses, strong beef-tea, jellies, and alcohol must be given, remembering from what has been said above, that a red glazed tongue and delirium are not contra-indications to the use of the last-mentioned.

FEBRICULA.

Lat. *Febricula.* Fr. *Fièvre éphémère.* Ger. *Febricula.*

DEFINITION.—Simple continued fever, non-contagious, occurring sporadically, of not more than three or four days' duration, and having no obvious distinguishing character.

Syn. EPHEMERAL FEVER, SIMPLE CONTINUED FEVER.

A simple fever, generally lasting from one to four days, and to which the term *ephemeral* is very properly applied. It may, however, continue in some cases for nine or ten days. Ephemeral fever appears to be caused by over-exertion, errors in diet, or exposure to the sun's rays. It is not infectious, and unless complicated with other diseases, is rarely fatal. Very mild attacks of typhus or typhoid fever may be mistaken for this fever. It most frequently attacks young adults.

Symptoms.—Febricula, as a rule, sets in suddenly; but in some cases, for three or four days before the fever is developed, lassitude, anorexia, and a general feeling of discomfort may exist. The febrile movement is often intense, the heat of the body rapidly rising in a few hours from the normal temperature 98·4° to 104° F., and then as rapidly falling. The pulse varies from 100 to 120, or even 130 beats a minute. A hot skin, furred tongue, quick, full, and often firm pulse, frontal headache, pains in the loins and limbs, and complete loss of appetite, form the most prominent symptoms when the fever has passed the stage of incubation. Delirium may occur. The bowels are usually confined, the urine has a high specific gravity

(1035–1037), is small in quantity, and deep-coloured. Herpetic vesicles often occur about the mouth; but there is *no characteristic* eruption. The feverish symptoms may all subside in twelve, twenty-four, or thirty-six hours; but sometimes, however, the fever does not disappear for seven, eight, or ten days.

Copious perspiration generally attends a cessation of the fever; but in some cases hæmorrhage from the nose, uterus, or bowels, may also occur. Nausea and vomiting, or a large deposit of lithates in the urine, may also usher in the termination of the fever. It may be distinguished from typhus or typhoid fever by the abruptness of the attack, and by the existence of evident and ordinary causes, but it must be admitted that the diagnosis is in many cases at the outset impossible; still some assistance may be gained by noting the intense severity of the early symptoms as compared with the more gradual progress of typhus and typhoid fevers. In this intensity it closely resembles relapsing fever, but the jaundice, enlarged spleen and liver, and severe pains in the muscles and in the joints, present in relapsing fever, are here absent. Caution must be observed in not too readily attributing the fever to a local lesion; the sudden disappearance of the fever will correct this view.

Treatment.—The treatment in England consists in emptying the stomach by an emetic if necessary, and by the administration of saline purgatives to lessen the violence of the arterial excitement. Rest in bed and a low diet are indicated. The thirst may be allayed by cooling drinks, and complications, as they arise, treated on general principles. In treating the ardent fever of the tropics, venesection, leeches, and cold affusion to the head, and a free administration of purgatives and diaphoretics, are recommended. Murchison says, "I am inclined to think that life is often sacrificed by adopting less active means." During convalescence tonics and a nutritious diet must be given.

TABLE SHOWING POINTS OF DISTINCTION BETWEEN RELAPSING, TYPHUS, AND TYPHOID FEVERS.

	RELAPSING FEVER.	TYPHUS.	TYPHOID.
Duration...	Seven-day fever. Relapses occur on the 14th day from the invasion ; the 7th from the critical sweating.	Fourteen-day fever.	Twenty - one day fever.
Mortality...	About 4 per 100.	1 in 5 or 6.	1 in 6.
Age..........	All Ages.	Adult life.	Youth and adolescence.
Tendency to occur...	Epidemically.	Epidemically.	Endemically.
Anatomical Signs.....	Characteristic eruption absent. Petechiæ present differing from typhus by not appearing in the centre of exanthematous spots.	Mulberry rash. Petechiæ present and persistent.	Rose - color'd rash, congestion, and ulceration of Peyer's patches.
Diagnosis...	Muscular pains, roseola absent, herpes facialis. Tenderness and enlargement of liver. Sudden cessation of fever. Delirium rare.	Herpes facialis absent. Active delirium frequent. Peculiar dusky coloration of skin.	Roseola present. Diarrhœa present. Low muttering delirium.

TABLE—*Continued.*

	RELAPSING FEVER.	TYPHUS.	TYPHOID.
Cause.......	Starvation and over-crowding.	Over-crowding, *ochlesis*, and starvation. Contagion, miasm. Concentrated exhalations from human bodies.	Miasma, foul drinking-water, etc.

RELAPSING FEVER.

Lat. *Febris residiva.* Fr. *Fièvre à rechutes.*
Ger. *Typhus recurrens.*

DEFINITION.—A continued fever of short duration, characterised by absence of eruption, and an abrupt relapse occurring after an interval of about a week. One attack does not appear to confer immunity from subsequent attacks. If such immunity exists it must be of short duration.

FAMINE FEVER—SEVEN-DAY FEVER—BILIOUS REMITTENT FEVER—SPIRILLUM FEVER.

The above-mentioned names have been given to a contagious continued fever of short duration, the febrile phenomena abruptly terminating, and recurring after an interval of a week of comparative convalescence. A second, third, and even a fourth relapse may take place. The fever may be divided into four stages—(*a*) primary paroxysm; (*b*) intermission; (*c*) relapse; (*d*) convalescence. Relapsing fever is seldom fatal; when so, death may be due to syncope or to uræmia, but more frequently to some complication, such as dysentery or pneumonia.

Etiology, etc.—Extreme poverty and exhaustion following on long and continued privation, together with over-crowding, appear to favour the origin and spread of this fever. It affects all ages and both sexes indiscriminately, and may occur at any season of the year. As a rule, only the destitute are attacked, and when it spreads beyond these, direct communication with the sick appears necessary. Free ventilation, and freedom from direct contact with the sick, prevent the spread of relapsing fever. It may be communicated by the clothes of those attacked, and it has been known to immediately follow exposure to the poison; but, as a rule, the period of incubation varies from four to sixteen days. According to Murchison it can be generated *de novo*, and he gives as a strong argument in favour of this view, that it may suddenly reappear after years of entire absence. Sir R. Christison speaks of its "spontaneous generation" from "penury pent up in airless dwellings" as beyond doubt. In short, its chief, if not only cause, is starvation. Lebert maintains that a protomycetic origin to this fever has been most positively established. It is stated that in this fever, and in the splenic fever among the lower animals, bacteria (spirilla, more correctly spirochætæ) have been found in the blood, their presence being most marked during the febrile stage. The spirilla are held to constitute the contagious elements of the disease. They do not appear to be capable of resisting high temperatures; it is, therefore, probable that their constant appearance during the attack is due to successive generations.

Symptoms.—The invasion of the fever is sudden; chilliness, followed by severe rigors, pains in the head and limbs, usher in the attack. The patient takes to his bed at once, not so much from weakness, as in typhus, but because he feels "giddy." The temperature of the surface becomes intense, the thermometer marking as high as 102° to 107° F. A rapid rise in temperature to 106° or 107° F., commencing before the premonitory rigor, and before the pulse shows signs of quickening, and reaching its height just before the critical sweat, with a rapid fall to below the normal 98° F., is pathognomonic of relapsing fever. This subnormal fall in the temperature (96° to 92° F.), sometimes for a few hours rising above that of health, and then becoming normal till the relapse sets in, may assist the diagnosis. The rise in temperature is no sign of danger. The tongue, usually moist, is coated with a white fur, the edges and tip being red. The pulse is very rapid, from 110 to 140 beats a minute, but this frequency does not itself betoken increased danger. A systolic bellows murmur, probably a blood murmur, loudest at the base of the heart, is often heard. The respiration is quickened. There is intense thirst, and the appetite may be either absent or voracious. The face expresses pain, is flushed, and the eyes red, but the dingy colour of the skin common to typhus, or the circumscribed flush of typhoid, is absent. The skin in some cases has been described as bronzed. In fatal cases the face assumes a dingy purplish hue.

Vomiting of a bilious, greenish-yellow fluid, or of dark grumous matter, occurs in most cases. The bowels are constipated, and there is severe pain felt at the epigastrium. The urine is high-coloured, decreased in quantity, but varies with the fluid taken. The urea is increased during the paroxysms, and diminished during the interval. About the fourth day of the fever, jaundice, an unfavourable symptom, more or less intense, may occur; but this state is not attributable to occlusion of the gall duct. The liver and spleen are enlarged, and more or less tenderness over them is felt by the patient. There is no characteristic eruption, although in some cases small roseolar spots, or a reddish mottling, disappearing on pressure and never becoming petechial, has been observed. Petechia may occur, but do not betoken increased danger. Desquamation may follow an attack of the fever. The prostration is not so great as in typhoid fever. Want of sleep is a frequent and pressing symptom. Delirium seldom occurs, the mind usually remaining clear. When, however, delirium is present it generally occurs before the crisis, and is probably the result of the increase of urea or disintegrated nitrogenous materials in the blood. The urine before the delirium is scanty. About seven days from the beginning of the fever, the patient has a critical sweat, from which he emerges weak but convalescent, and joyful in the hope that he has now passed the worst; suddenly, however, on the seventh day from the critical sweat, and fourteenth from the first invasion of the symptoms, all his troubles return, and a well-marked relapse occurs. The critical sweat may take place on the third, or be delayed to the tenth, day of the fever. In some cases the critical sweat is replaced by diarrhœa or hæmorrhage from the nose, bowels, etc. The relapse may last four or five days, sometimes longer. Two or three relapses may occur before the patient regains his health. When death occurs it may take place suddenly, and without previous warning of danger.

Pregnant women affected with this fever, as a rule, abort—the women as a rule, however, recover.

Diagnosis and Prognosis.—Do not mistake this fever for typhus, typhoid, febricula, ague, yellow fever, incipient small-pox, bilious headache, and cerebral diseases, especially among children. The sudden invasion, the absence of a rash, and the well-marked relapse, serve to distinguish this fever from typhus or typhoid. It must, however, be borne in mind that epidemics of typhus and relapsing fever may co-exist, and that over-crowding and destitution favour the origin and spread of the former, whilst the origin of the latter appears to be due to destitution, and its spread to over-crowding. In co-existing epidemics, typhus generally prevails at the end, and relapsing fever at the onset of the epidemic. An attack of typhus does not confer immunity from relapsing fever, and, as a rule, typhus occurs but once, whereas relapsing fever may occur *several times* in the same individual. In nearly all the observed cases the attack of relapsing fever preceded the typhus attack. The prognosis is, on the whole, favourable, but the disease appears to be more fatal among the black than the white races.

Treatment.—The treatment should consist in the exhibition of a sharp purgative or emetic at the outset of the disease. Rest in bed, and a diet suited to the condition of the patient, should be allowed. Administration of the mineral acids has been highly recommended. The action of the kidneys, by small doses of nitrate of potash and saline diuretics, must be carefully maintained, to prevent the tendency to uræmic poisoning, F.1. The ophthalmia, which often occurs as a sequel, is best treated by bleeding; two or three leeches applied to the temples will suffice. The pupil must be kept dilated with atropine. Frequent sponging of the skin with tepid water or water and vinegar is a source of great comfort to the sufferer. Other symptoms, as they arise, must be treated as the occasion requires. Lebert is of opinion that relapsing fever may be treated quite safely in a general ward of a hospital. He, however, recommends free ventilation and perfect cleanliness in the wards. Typhoid fever may be so treated, but the exclusion of typhus is imperative. Quinine and digitalis have not been found of use in the treatment of this fever.

Geographical Distribution.—Relapsing fever is indigenous to Ireland. Epidemics have occurred in the British Isles, and also in Poland, Germany, and Russia. It has on various occasions been introduced into America, but has not spread in that country. It has been noticed occasionally in India, in Egypt, and Algeria. It has probably been confounded with tropical yellow fever, or bilious remittent fever of malarious origin.

TYPHUS FEVER.—TYPHOID FEVER.

Before entering upon a discussion concerning the nature of two of the most important continued fevers, *typhus* and *typhoid*, it may be as well to place in parallel columns a list of the principal points in their symptomatology and pathology, which may help to distinguish them:—

TYPHUS	TYPHOID
attacks persons of both sexes and all ages indiscriminately, and is generally traceable to contagion. The invasion is sudden; the head symptoms in the course of the fever are severe, delirium with contracted pupils usually occurring towards the end of the first week. The eruption, which belongs to the class *maculæ*, appears on the skin in the form of a mulberry-coloured rash, between the third and seventh day of the disease, imperfectly disappearing on the application of pressure, and continuing until the termination of the fever. The rash is seen all over the body, but seldom appears on the face. The spots may become petechial. Diarrhœa is seldom present during the course of typhus, and hæmorrhage from the bowels never occurs.	chiefly occurs in the young, from twelve to twenty-five years of age, and but rarely after forty. It is during this period that the solitary and aggregate glands of Peyer are most fully developed. As age advances, these glands disappear, so that after forty-five only traces of their previous existence can be discovered. The lesion in these glands forms the "anatomical sign" of typhoid. The invasion is insidious, the premonitory symptoms lasting about a week. The abdominal organs are much affected; congestion and ulceration of Peyer's patches occur, with attendant diarrhœa; and, in about one case in every three, there is more or less hæmorrhage from the bowels. The abdomen is tense, and pain is felt on pressure. There is low muttering delirium, with dilated pupils. The eruption, which belongs to the class *papulæ*, appears about the seventh day, and is chiefly found on the chest and abdomen. It comes out in successive crops of rose-coloured, slightly-elevated lenticular spots, in size somewhat less than a split pea. The rash disappears on pressure. In two or three days the spots fade away, to appear again on another part of the body. "This successive daily eruption of a few small very slightly elevated rose-coloured spots, disappearing on pressure, each spot continuing visible for three or four days only, is peculiar to, and absolutely diagnostic of, typhoid fever."

Typhus prevails most frequently among the poor, and is a disease of adult life. It is due to bad diet and over-crowding. The disease runs its course in from fourteen to twenty-one days, and is most dangerous about the end of the second week. Typhus fever is most frequently fatal before the fifteenth day, never after the twentieth. The tendency to death is by coma.	The average age of mortality appears to be about twenty-three. The rich are most frequently attacked. The miasma arising from the decomposition of animal matter, or the pollution of drinking water by drains or sewers, is the fertile source of this fever. It is not contagious. Pregnancy is said to give almost entire exemption from typhoid. Relapses are not uncommon in typhoid. The duration of the fever is more or less prolonged, from three to six weeks, but its maximum of intensity is reached about the third week. Typhoid is more frequently fatal after the twentieth day; more than half the deaths occur after this period. The tendency to death is by asthenia.

TYPHUS FEVER.

Lat. *Typhus.* Fr. *Typhus.* Ger. *Exanthematischer typhus.*

Definition.—A continued fever, characterised by sudden accusion, great prostration, and a general dusky, mottled rash, without specific lesion of the bowels.

JAIL FEVER—SHIP FEVER—CAMP FEVER—HOSPITAL FEVER—OCHLOTIC FEVER, ETC.

Typhus fever has been long known, having been described by Fracastorius and Cordanus, but it has only in recent times been distinguished from typhoid fever, with which it was confounded; an error in diagnosis that has resulted in much bitter discussion and little progress. It is a contagious continued fever, having a duration of from fourteen to twenty-one days, attended with great depression of the vital functions. The tendency to cerebral complications is great, hence this disease has been called by some *brain fever.* Dr C. Murchison has suggested that epidemic cerebro-spinal meningitis is typhus complicated with inflammation of the brain and membranes. The above-mentioned authority maintains that plague is perhaps the typhus of warm countries. Bubonic typhus, or typhus complicated with buboes, etc. Typhus is also characterised by the appearance, on or about the fourth or fifth day of the fever, of a peculiar maculated eruption on the skin.

Symptoms.—The invasion of this fever is in most cases *abrupt;* sometimes, however, gradual and obscure, one or two days of slight indisposition ushering in the attack. As a rule, however, fits of shivering attended with severe headache, pains in the back and limbs, mark the onset of an attack. Other symptoms, common to most acute diseases, now make their appearance. There is loss of appetite, the face is flushed and dusky, the eyes injected and watery, tongue furred then dry and feverish, the skin hot; there is great thirst, a great sense of chilliness, a full, quick, compressible pulse, constipation, scanty, high-coloured urine, and a sensation of intense lassitude and disinclination to move about. The demand for cold water is urgent. The muscular debility is usually so intense that the patient takes to his bed on the second or third day of the fever, and in most cases is incapable of moving from it without assistance. The expression of the face becomes dull, heavy, and stupid. From the fourth to the seventh day, usually on the fourth, the characteristic rash of typhus makes its appearance. The *maculæ*, *mulberry*, or *rubeoloid rash*, occurs under two forms—as distinct spots or as a sub-cuticular mottling. The spots first appear of a dusky pinkish-red colour, slightly raised above the skin, and disappear on pressure; but after the first and second day they become dark and more dingy, less elevated, and do not disappear on pressure, only becoming slightly paler. On the eighth or tenth day true petechiæ are in some cases formed. Petechiæ may be defined as minute purplish spots or sub-cutaneous ecchymoses, which do not disappear on pressure. Flea bites have been mistaken for the petechiæ of typhus; the former are more circumscribed, disappear on pressure, and have a central puncture which does not disappear when pressed. The rash is most abundant in severe cases, and never appears in successive crops as in typhoid fever. The rash varies in colour on different parts of the body, and gives a mottled aspect to the skin. It may be said to go through three stages:—

1. Pale dirty pink or florid, slightly elevated, and *disappearing* on pressure.
2. Reddish-brown or rusty, not elevated, and *slightly disappearing* on pressure.
3. Livid and petechial, *not affected* by pressure.

It first appears on the front of the axilla and sides of the abdomen, then on other parts of the body, and then subsides between the fourteenth and twenty-first days of the fever. The intellect soon becomes affected, and there is much confusion of ideas. About the seventh day, low muttering delirium occurs, "typhomania," or the patient may become excited and noisy, making frequent attempts to jump out of bed. Niemeyer mentions a case in which a patient was sent to him in a strait-jacket, from jail, suffering from typhus. During the early stages of delirium, the patient may be restored to consciousness by a sharp question; but as the fever progresses, he becomes less easily roused, more stupid, and his countenance wears a half-drunken expression, at no time betraying anxiety. The nervous excitement increases towards night, and the prostration towards morning. The surface of the body, particularly the face, assumes a dusky or dingy hue. As the disease progresses, the tongue becomes covered with a thick brown or black coating, sordes appear on the teeth, and the lips are dry and cracked. He is now unable to move himself, lies perfectly prostrate in bed, and picks at the clothes, entirely indifferent to everything going on around him, and his motions and urine are passed involuntarily. Diarrhœa, if present, is slight; and there is little, if any, abdominal tenderness, but sometimes slight tympanitis. There is usually more or less catarrh, attended with slight cough, proceeding from sub-acute bronchitis. The respiration is sometimes impeded and accompanied with frequent sighing, but as a rule it is increased, the average being about 21 a minute. The pulse is feeble and quick, reaching 110 to 120 beats, and is rarely dicrotinous as in typhoid. The temperature of the surface, as registered by the thermometer, varies from 102° to 106° F., being generally a degree or two higher in the evening than in the morning. It reaches its maximum between the fourth and seventh days, and permanently high morning and evening temperatures alone betoken gravity. The urea and uric acid are increased, whilst there is a gradual diminution of the chlorides. Albuminuria is also not unfrequent. About the fourteenth day sudden amendment in the condition of the patient may take place, a refreshing sleep stealing over him, from which he awakes conscious but fearfully weak. A rapid decline of temperature betokens the setting in of convalescence. In fatal cases death takes place about the twelfth or fourteenth day, the coma becoming more profound as death approaches. The odour from the body is often most offensive. Severe bed sores may form on the sacrum and hips; and the extremities, the nose, ears, etc., may become gangrenous.

Several complications may occur during the course of the fever; these are generally to be referred to the cutaneous, nervous, respiratory, circulatory, alimentary, and urinary systems:—

Cutaneous.—Among the true exanthematous fevers, the absence of a rash, as a rule, betokens a mild attack. The opposite is, however, the rule with typhus. In the exanthematous fevers the eruption soon disappears, in typhus it is persistent.

Nervous System.—Convulsions, due to the blood-poisoning, are fortunately of rare occurrence. Increased cerebral excitement, ending in delirium more or less severe, generally supervenes towards the end of the first week; in non-fatal cases, the delirium passes off in three or four days.

Respiratory System.—Pneumonia, generally in the lobular form, is not of unfrequent occurrence, sometimes ending in gangrene of the pulmonary tissue. Bronchitis may also be present, and the patient is frequently troubled by a dry cough, with slight expectoration.

Circulatory System.—Dr Stokes has described a peculiar form of cardiac lesion, which he has named "typhus softening of the heart." Sometimes the muscles of the heart are found to have undergone the waxy change described by Zenker as almost peculiar to typhoid. A diminution of the impulse, and an impairment or loss of the first sound, indicate the cardiac phenomena of typhus.

Alimentary System.—Purpuric extravasation found in the intestines, the walls of which are, as a rule, healthy; "the solitary and agminated glands may be a little enlarged, and the latter may present that dotted appearance which has been compared to a shaven beard." The liver is soft, and on section shows a characteristic dull clay-like lustre.

Urinary System.—The kidneys may be found enlarged, especially if death follows an attack of convulsions. The urine is sometimes albuminous.

The **prognosis** must be formed on a careful review of the symptoms, the age and condition of the patient being taken into consideration. Death generally occurs from a combination of asthenia and coma. Pregnant women do not, as a rule, abort.

ETIOLOGY.—1. PREDISPOSING. 2. EXCITING.

Predisposing Causes.—Poverty, over-crowding, with imperfect ventilation and unwholesome food, together with mental or bodily exhaustion, form the chief predisposing causes. Sex does not predispose to typhus, but with regard to age typhus is, as a rule, a disease of adult age, especially after thirty; hence it is a disease, the arrest of which is important to the State, as it slays the bread-earners, leaving widows and orphans dependent on the parish. It is also a disease of the poor. The seasons do not appear to influence its spread, epidemics occurring at different periods of the year. The temperature and hygrometric state of the atmosphere influence it but little. It may be said that, as a general rule, any cause which lowers the vitality of the individual, added to overcrowding—*ochlesis*—favours the origin and spread of typhus.

Exciting Causes.—The exciting cause is a specific poison generated by the bodies of persons crowded together and placed under improper sanitary and hygienic conditions. Typhus may be conveyed by those *not* suffering from the disease.

Treatment.—The treatment has now to be considered. Sydenham's remarks on the treatment of fever are worth recording here:—"I cannot bring my brain to comprehend the meaning of those physicians who are continually talking about the administration of remedies that promote the concoction of the febrile matter, points which they insist upon when called in at the beginning of the diseases. At the same time, they have no hesitation in recommending medicines that are intended to control the fever. Why! the fever itself is nature's instrument by which she separates the pure parts from the tainted ones!" He himself recommends the treatment now adopted, and best described as the "expectant treatment." The patient should be put to bed in a well-ventilated room. It is difficult to get too much ventilation for typhus patients. Free ventilation, as a rule, prevents the spread of the disease, and also, to a great extent, secures the safety of the attendants. Bed sores are best treated with hot linseed-meal poultices, sprinkled with fine charcoal. To prevent the occurrence of those troublesome complications, the parts resting on the bed, in cases where a water-bed cannot be procured, should be bathed with a strong solution of alum; it hardens the skin. If the case is seen early, an emetic may be given with advantage. Beware of doing too much. Cooling drinks to allay the thirst, and tepid sponging of the body, together with hygienic and supporting measures, will form the most important part of the treatment of typhus. Be careful also in the use of alcoholic stimulants; do not, because it is a case of fever, rush to the brandy bottle; and remember that the occurrence of delirium does not always prohibit the use of alcohol, the delirium often being due to extreme prostration. When shall we use stimulants? As a rule, wait till the second week of the fever. But if at any time before or after that period there is marked exhaustion, the first sound of the heart becoming obscure, with a feeble pulse, stimulants may be then given, beginning with a table-spoonful of brandy or whisky, carefully watching its effect. Continue the administration of the alcohol if you find "that it agrees with the patient." Dr Haughton maintains that "the effects of alcohol administered in fever, when the temperature does not exceed 105° F., are twofold—immediate and secondary. The immediate effect is to supply a hydro-carbon to the blood, which is decomposed by it in preference to the body tissues. The secondary effect of alcohol is to change the blood itself, which thus loses its oxidising qualities, in consequence of which the temperature falls, the hyperdicrotic character of the pulse disappears, and the distinctive metamorphose of the tissues becomes lessened." Increased febrile movement and restlessness, with delirium, suggest the discontinuance of the alcohol. Tonics and change of air will be required during convalescence.

TYPHOID FEVER.

Lat. *Febris enterica.* Fr. *Fièvre typhoïde.* Ger. *Abdominal-typhus.* Syn. *Ileo-typhus.*

Definition.—A continued fever, characterised by the presence of rose-coloured spots, appearing in successive crops, chiefly on the abdomen, with a tendency to diarrhœa, with specific lesion of the agminated and solitary glands of the intestines. The period of incubation is from six to twenty-four days or even longer. Enteric fever occurring in the child is often named infantile remittent fever.

Enteric Typhus—Pythogenic—Abdominal Typhus.

Typhus and typhoid fever were long confounded together. They differ in the following particulars:—

Typhus fever is a purely infectious disease; typhoid belongs to the miasmatic-contagious diseases.

Typhus can be transmitted directly from person to person; its contagion is as intense and evident as is that of the acute exanthemata. Typhoid fever, on the contrary, is never transmitted from person to person *(Liebermeister).*

Symptoms.—During the premonitory stage of typhoid fever, the patient is chilly, indisposed to exertion, and hangs over the fire. His limbs tremble, he complains of feeling sick, with entire loss of appetite. Pains in the limbs of a rheumatic nature are complained of, and he may suffer from repeated attacks of hæmorrhage from the nose. His breath becomes offensive, his throat sore, his bowels usually irritable, sometimes confined, his sleep broken, his pulse quickened, his tongue white; he daily becomes weaker, and at last has probably a violent rigor, followed by great heat of the skin and intense headache, and such muscular debility that he takes to his bed. This is the accession, and the course of the fever has now set in, and may be divided, for the sake of convenience, into three weekly periods:—

First Week.—In the first week, the leading characters are vascular excitement and nervous depression. The fever during this period shows a gradual and steady increase.

There is a frequent and bounding pulse of from 90 to 110, keeping pace more or less with the rise in temperature, sometimes dicrotinous. There is much heat of skin, the temperature varying between 96·8° and 104° F., great thirst, and at night there is delirium and great restlessness. The febrile movement is the most important symptom in determining the diagnosis, prognosis, and treatment. The fever in great measure commands the situation *(Griesinger)*. The sleep is broken by frightful dreams, the patient often muttering, or uttering in a loud voice, portions of detached sentences. When awake, he lies with his eyes half open, quite conscious, but showing little interest in what is going on around him. In the course of the delirium, the patient may be temporarily restored to consciousness by the entrance of a stranger into his room, or by a sharp question addressed to him.

The oppression about the mental faculties is peculiar, and the patient can only with difficulty be induced to give an account of himself, or indeed to complain of anything but his head.

The tongue at first is but slightly coated with a white fur, but appears moist and broad, and is marked by the teeth. The epithelial covering generally falls off, leaving "a moist, red, smooth tongue, that looks as if covered with goldbeater's skin, or else is already inclined to dryness. If there be at first a thick adherent coating on the tongue, it is usually detached from the point posteriorly, and from the sides toward the middle, so that the whitish-yellow coating appears enclosed in a very red border, which constantly increases in width; but, in some cases, the detachment begins centrally, so that in the middle of the tongue there is a red stripe that has a peculiar tendency to become dry, and at the sides two whitish-yellow, moist, slimy stripes. The central stripe is often broad anteriorly, and disappears posteriorly, so that on the point of the tongue we see a red triangle, with the apex posteriorly."

The bowels may be confined at the outset of the fever; but towards the end of the first week, the characteristic diarrhœa sets in, consisting of light-coloured, semi-fluid, yeast-like or pea-soup stools, of which he may have, perhaps, seven or eight in the twenty-four hours. The abdomen feels full and tense, and there is slight tenderness felt on firm pressure made, especially over the right iliac region. A gurgling sensation—ileo-cæcal gurgling—is also experienced by the fingers.

The spleen becomes enlarged towards the end of the first week, being sometimes two or three times its natural size, due to increased vascularity and the production of lymphatic elements, but owing to its softness, and to its displacement by the distended intestines, its outline can only be defined with difficulty.

Emaciation rapidly sets in, the muscles wasting as well as the fat. The urine is scanty, high-coloured, and loaded with urates, with marked diminution in the quantity of the chlorides. The diminution in the chlorides may be in part due to the spare diet, or to their elimination in large quantities with the stools or sweat.

Second Week.—During the second week, the fever is continuous; the rose-coloured eruption now makes its appearance in successive crops, and is chiefly found on the chest and abdomen. Each crop lasts three or four days. If the finger be passed *very* lightly over the surface of the body, the eruption can be detected as slightly-elevated soft pimples, unlike the hard "shotty" feel presented by the pimples in the early stage of the small-pox eruption. The eruption disappears on pressure, and when it fades away no scar is left. The liability to *sudden relapses*, attended with the appearance of new crops of the eruption, is very characteristic of typhoid fever. The headache becomes less, the pallor of the face greater; increasing emaciation and weakness, a greater tendency to lie on the back, a listless stupor, the voice weaker, and the speech often unintelligible, together with calm or furious delirium, and coma vigil, mark the second period. Towards the close of this period the tongue becomes dry, sordes form on the teeth, the dejections fœtid, often passed involuntarily, sometimes containing blood, and also at times retention of urine. There may be a slight cough, sibilant râles, and some amount of hypostatic pneumonia. The fever is still continuous, the temperature vacillating between 102·2° and 104° F.

Third Week.—During the third week the fever becomes gradually intermittent, and the symptoms either slowly decrease in severity or are increased; in the latter case the emaciation and debility become extreme. The patient lies extended on his back, sinking towards the foot of the bed, making no effort to change or preserve his position. The pulse is quickened and irregular, the respiration more difficult, and severe and debilitating sweats now occur. There is a purplish flush on his cheeks, and sordes cover his gums and lips. His tongue is dry, shrunken, stiff, and black, like a bit of leather; his urine, from inaction of the bladder, is retained, and his fæces pass involuntarily. An alarming symptom at this stage is to find the patient lying helpless at the lower part of the bed.

The noisy delirium now gives place to increasing stupor; he no longer recognises his friends, but mutters incoherently, or picks vacantly at the bed-clothes, and if he recovers, will not remember anything that has occurred during his illness. An erythematous rash in most cases appears on the sacrum, the cutis becomes exposed, and bed sores supervene. At length, and sometimes suddenly, as if by a crisis, he begins to improve; the tongue becomes moist at the tip and edges; there is some return of reason, like that of a little child; he asks for food in a ravenous way, and expresses a desire to get up.

Still, however, there is a stage of sequelæ, in which he suffers from internal mischiefs that have already taken place, and from one of which he may even yet be carried off. Among these complications may be mentioned, peritonitis from perforation of the intestines, bronchitis, pleurisy, pneumonia, erysipelas, mumps, hæmorrhage from the bowels, ulceration of the larynx just below the vocal cords. Wilks describes a case in which the ulcer in the larynx "led into a space between the trachea and œsophagus, through which the air had escaped, and had given rise to general emphysema." A complication like this may give rise to the emphysema that has been sometimes noticed during the course of typhoid fever. It is at the end of the third week that death most frequently occurs. The bowel affection may itself prove fatal, either in an early stage by hæmorrhage from an ulcerated vessel, or in the convalescent stage, about the fifth or sixth week, by perforation of the bowel and escape of its contents, causing peritonitis. This is to be feared when, after an attempt at recovery, the patient still suffers from irritable bowels, with occasional hæmorrhage, the tongue remaining preternaturally red at the tip and edges and the pulse frequent. When perforation takes place, the abdomen suddenly swells and becomes excessively tender, the patient generally dying in three or four days. Such is a brief account of a typical case of typhoid fever.

Pathology, etc.—The anatomical characters peculiar to typhoid and found after death, are seated in Peyer's patches, and in the solitary glands of the small and large intestines. The glands are found enlarged, congested, and studded with ulcers, varying in size.

Changes found in the Glands—

1. Increased vascularity.
2. Proliferation of the lymphatic contents.
3. In colour they are greyish white or reddish.
4. Contents cellular, soft, and brain-like.

Terminations—

A. Resolution. *B*. Ulceration. *C*. Cicatrisation.

Ulceration—

(*a*) One large ulcer, size of gland, or several small ones.
(*b*) Edges formed by inflamed mucous membrane, ragged and undermined.
(*c*) Base smooth, formed by coat of intestine.

It may be as well here to give the signs which distinguish the typhoid intestinal ulcer from the tubercular:—

Typhoid Ulcer.	Tubercular Ulcer.
1. Long diameter parallel to long axis of the intestine.	1. Long diameter transverse to long axis of the intestine.
2. Edges thin, ragged, and undermined.	2. Edges thick, undulated, indurated, not undermined.
3. Does not lead to stricture of the intestines.	3. Often the precursor to stricture.
4. As a rule heals readily.	4. Seldom heals.
5. Seldom accompanied with local or general peritonitis.	5. As a rule accompanied with local or general peritonitis.
6. Floor smooth.	6. Floor rough and nodular.
7. Perforation common.	7. Perforation rare.
8. Limited to Peyer's patches.	8. Not limited to Peyer's patches.

Other lesions are also present; the spleen may be more or less enlarged and softened, the walls of the heart flaccid, and the muscular tissue softened; or the muscular structure may have undergone the change described by Zenker, in which the muscle in parts becomes too solid, is easily torn, has a peculiar waxy appearance, and glistens when cut into.

The blood found in the cavities of the heart is of a dark, venous colour, and imperfectly coagulated. The kidneys are sometimes greatly congested, enlarged, and the uriniferous tubes filled with exfoliated epithelium; at other times they are unusually pale. The lungs are gorged with blood of a dark colour, and present a spotted appearance both externally and internally *(Wilks)*. Consolidation is found at the posterior edge of the lung, seldom passing beyond the stage of red hepatisation. The pneumonia is lobular, patches of the lung being attacked. The mesenteric glands are also enlarged and softened. The membranes of the brain may or may not be inflamed and congested, the delirium being no index of the inter-cranial disturbance.

The gradual rise in temperature during the first three or four days in typhoid fever is very characteristic—thus, on the

Morning of the	first day,	98·6° F.;	evening,	101·3° F.
"	second day,	100·22° F.;	"	102·56° F.
"	third day,	101·66° F.;	"	103·64° F.
"	fourth day,	102·56° F.;	"	104·54° F.

It will be seen from the above table that every evening the temperature is about 2° F. higher than in the morning, and in the morning 1° F. lower than the previous evening, till on the fourth evening a temperature of 104° or 104·52° F. is reached.

Typhoid may be excluded—

1. If the temperatures on the evenings of the second, third, or fourth days are only approximately normal.
2. If the temperature on the first three evenings be the same.
3. If the temperature on two of the first three mornings be the same.
4. If the temperature on the first two days rises to 104° F.

The temperature during the latter part of the first week, and the early part of the second, is almost uniform. The maximum is reached on or about the fourth day, and then remains stationary for a day or two, decided defervescence not taking place till the twenty-first day.

During the third week the temperature shows a marked difference from that of typhus. In typhus the normal temperature is regained about the fourteenth day; in typhoid, the fever is continued into the third week. When the temperature for two successive evenings has closely approached to normal, convalescence has set in.

Etiology.—Typhoid fever appears to arise from the emanations of foul drains and cesspools, and is considered by some to proceed from a special poison contained in the excreta, and which is disseminated by the effluxion from water-closets, privies, and drains. The prevailing opinion at present is that typhoid fever is due to a specific germ, and is not generated by the mere decomposition of animal excreta. It occurs most frequently in the months of October, November, September, and August. The observations of Pettenkofer on the wells of Munich suggested to Buhl the probable relationship between the height of the ground water and epidemics of fatal typhoid fever, the outbreaks occurring when the ground water was lowest, but especially when it had reached an unusual height and had then rapidly subsided. This view, although confirmed by subsequent observations in Germany, has not been accepted in England, where impurity of the drinking water is generally accredited with the causation of the outbreaks. Pettenkofer considers the following conditions to be necessary for the production of typhoid fever:—(*a*) Unusual height of ground water, followed by rapid sinking. (*b*) Impurity of the soil from animal impregnation. (*c*) Heat of the soil. (*d*) Presence of a specific germ.

Diagnosis and Prognosis.—A careful consideration of the symptoms and course will distinguish it from typhus. In the early stages of mild cases the disease may be mistaken for a somewhat severe form of gastric disturbance with dyspepsia. The prognosis will depend upon the duration of the fever and the absolute height of the temperature. Patients when not dying from perforation of the intestines may die from paralysis of the heart or brain, the result of long-continued high temperature. The greater the daily fluctuations, and the presence of even well-marked remission of the pyrexia, add greatly to a favourable prognosis. A strong, not too frequent, pulse betokens no immediate danger, and, other things being equal, is decidedly in favour of the patient. Constant and prolonged cerebral disturbances are signs of the utmost gravity.

This disease must not be confounded with simple fever or with peritonitis.

Treatment.—The most important part of the treatment of typhoid fever depends upon hygienic and supporting measures. The patient should be placed in a large, well-ventilated room, from which all unnecessary articles of furniture have been removed. If seen during an early stage of the disease, a hot bath will often afford great comfort to the patient. The use of the cold bath is strongly recommended by many German physicians, and is certainly of use when the temperature is persistently high and prolonged. In severe cases Leibermeister recommends that the baths should be repeated every two hours, night and day if necessary, so that twelve baths be given in the twenty-four hours, and in some instances more than two hundred baths have been administered during an attack of the fever. Any temperature in the rectum above 104·5° F. indicates the necessity for a bath. The method adopted has been before described. The diet should consist of milk, beef-tea, soups, and light boiled eggs. Alcoholic stimulants are indicated if the pulse becomes feeble, small, and compressible, but in the early stages they are not required. Calomel has been recommended in the reatment of this fever. "I have given this medicine, with but few exceptions, to every case of the fever admitted before the ninth day of the disease. I usually give three or four eight-grain doses during the first twenty-fours hours. After having employed this method now on about 800 patients, I still feel that I have every reason to continue it, and to recommend it to others" *(Leibermeister)*. The same authority recommends the use of quinine in doses so large that from twenty-two to forty-five grains be taken "*within the space of half-an-hour or, at most, an hour.*" This dose is not repeated for twenty-four hours, in most cases not for two days. Quinine is rapidly expelled by the kidneys, so that to produce its antipyretic action it must be given in large and frequent doses, long intervals in its administration lessening its value for this purpose. As an antipyretic, digitalis may be given, but care is required, and the more frequent the pulse the greater the caution. A mixture containing chlorate of potash and syrup, acidulated with hydrochloric acid, may be given, and was the chief medicine I employed during a severe epidemic of typhoid, and with the best results. The diarrhœa, if excessive, may be checked by the administration of the chalk mixture, or lime water. In any case if the bowels are moved more than twice in twenty-four hours, attended with great prostration, astringents should be given—the mineral acids to be preferred. Never be in a hurry to give aperients because patients wish it. As both typhus and typhoid run a definite limited course, the object of the physician is to keep the patient alive till the disease has worn itself out, therefore some of the heroic German methods should only be adopted in very rare cases. The bladder should be examined daily, and a catheter used if necessary. In the convalescence of typhoid fever, be very careful that solid food be not too soon taken; remember the tender ulcerated condition of the intestinal mucous membrane, and the liability to perforation.

INFANTILE REMITTENT FEVER.

Lat. *Febris infantium remittens.* Fr. *Fièvre rémittente des enfants.* Ger. *Abdominal-typhus im kindesalter.*

Definition—As Typhoid.

The true pathology of this disease was long unknown, till the researches of Dr Charles West proved its identity with typhoid fever.

Symptoms.—The disease may occur in two forms, a mild and a severe; in the former, the symptoms come on gradually and imperceptibly; in the latter, the invasion is sudden and the severity marked from the first. The child in the milder form appears ailing for some days, then there is loss of appetite and troublesome thirst; in the day-time he is listless and fretful, and towards evening drowsy and feverish, and the night is passed in a restless and uncomfortable slumber. Sometimes there are profuse sweats, alternating with dryness of the skin, but no relief is afforded to the child by their recurrence. The bowels at first may be confined and then relaxed, or the relaxation may be contemporaneous with the accession of the disease. The stools are unhealthy, very offensive, and of a peculiar yellow-ochry colour, and separate on standing into a supernatant fluid and a flaky sediment. The tongue is red, dry, and coated in the middle with a whitish fur, through which the enlarged papillæ are to be seen projecting. The belly is soft, and slightly painful when pressed. Sometimes when the child is visited in the morning, it is found sitting up trying to play with its toys, but indifferently pleased at best and requiring constant change. Towards evening all the feverish symptoms return, and these remissions may occur for several days. The pulse is quick, and there is slight cough.

The above constitute the symptoms of the accession, and to the end of the first week.

During the second week the nights are more restless, the eyes during sleep are half open, the thirst is greater, and some amount of delirium is often present. Sometimes also, during this period, an exacerbation of the fever occurs about eleven o'clock in the morning, but as the disease abates, the morning attacks cease to recur. The rose-coloured spots, so characteristic of typhoid fever, if present at all, make their appearance towards the end of the second week.

During the third week the severity of the symptoms abates, or, becoming more severe, the case ends fatally. As a rule, however, the severer form of the disease gives some indication of its severity from the very first. The temperature is then high, sometimes reaching 105° F. There may be certain chest complications which will require attention.

Dr West says that, in his experience, typhoid fever in children seldom terminates fatally, and death but rarely follows from those complications so fatal to adults.

Typhoid fever in children must not be confounded with gastric derangement from errors of diet, dentition, etc. The first is more frequent in boys than girls, and is rare before five years of age. The severity and continuance of the symptoms in typhoid must also be considered. The diagnosis from acute tuberculosis is more difficult.

Treatment.—The treatment is to a great extent the same as in typhoid in the adult. A simple saline draught, the citrate or chlorate of potash in syrup and water. If aperients are necessary, senna or castor oil may be given. The diarrhœa may be checked by chalk mixture or hydrargyrum *cum* cretâ, and Dover's powder. A nourishing diet—milk, beef-tea, and mutton broth, and wine if necessary. I have found that beef-tea in some cases increases the diarrhœa, when this occurs a change to chicken or veal broth has been most serviceable.

TABLE GIVEN AS AN AID TO DIAGNOSIS.

REMITTENT FEVER OR TYPHOID.	ACUTE HYDROCEPHALUS	PNEUMONIA.
1. Rare before five years of age, and seldom if ever met with under three.	1. Most frequent under five years of age.	1. May attack children at any age.
2. Vomiting most frequently absent even at the commencement of the fever; absence of nausea.	2. Vomiting most frequently present; constant nausea.	2. Vomiting slight, and soon ceasing; no nausea.
3. Bowels relaxed even at the beginning of the attack; evacuations watery; fæces of a lightish yellow.	3. Evacuations scanty, dark, or mud-coloured; deficient in bile.	3. Evacuations natural, but in exceptional cases there may be diarrhœa.
4. Tenderness over the abdomen, greatest in the iliac regions; flatulence well marked.	4. Absence of abdominal tenderness.	4. Tenderness over the abdomen misleading, for children may mean the chest when they say the belly.
5. Tongue with a thin covering of yellow fur in the centre and root, tips and edges red and dry from almost the beginning of the fever.	5. Tongue moist and coated with a white fur in the centre.	5. Tongue redder than in hydrocephalus.
6. Distaste for food, but constant demand for drink, especially cold water.	6. Distaste for drink and food.	6. There is often great thirst.

TABLE—*Continued.*

REMITTENT FEVER OR TYPHOID.	ACUTE HYDROCEPHALUS.	PNEUMONIA.
7. Skin very hot, and pungent.	7. Dryness of the skin, but not much heat.	7. Skin hot and dry.
8. Seldom complains of its head ; delirium is of early occurrence ; hydrocephalus may however supervene.	8. Child complains incessantly of its head, true delirium seldom occurring till near the fatal termination.	8. Some slight pain in the head.
9. Remissions — better in the morning, worse at night.	9. Symptoms fluctuate, but no *definite* periods at which the symptoms invariably remit, or are increased in severity.	9. No remissions.
10. Absence of signs of pneumonia on auscultation till the fever has continued for some time.	10. Absence of chest complications.	10. Auscultatory signs of pneumonia.

CEREBRO-SPINAL MENINGITIS.

Lat. *Febris cerebrospinalis.* Fr. *Fièvre cérébrospinale epidémique.* Ger. *Epidemische meningitis genickkrampf.*

Syn. *Malignant Purpuric Fever—Epidemic Cerebro-spinal Meningitis.*

DEFINITION.—An infectious malignant epidemic fever, attended with painful contraction of the muscles of the neck and retraction of the head. In certain epidemics it is frequently accompanied with a profuse purpuric eruption, and occasionally by secondary effusions into certain joints. Lesions of the brain and spinal cord and their membranes are found on dissection.

Inflammation of the membranes of the brain and spinal column sometimes appears as an epidemic. The name "spotted Fever" has also been proposed for this affection.

Symptoms.—In most cases the patient is suddenly attacked by headache, nausea, violent vomiting, and acute pain down the spine. The tongue may be either clean or furred, or, as the disease progresses, dry. Chills sometimes occur, as also vertigo and diarrhœa; but constipation is more frequently present. The urine is albuminous and may contain blood. The slightest noise or movement of the body greatly increases the pain. Tetanic spasms not unfrequently occur, and opisthotonos may even be present. The patient frequently holds his head back to relieve the pain in the neck. The temperature is seldom high. A herpetic eruption frequently breaks out round the mouth, but may also appear on the trunk or extremities. The eruption distinguishes this disease from other forms of meningitis. All the symptoms, delirium, sleeplessness, etc., which mark the presence of inflammation of the membranes of the brain and of the spinal cord, are present. Inflammation of the eyeball, ending in suppuration and entire destruction of the organ, sometimes occurs.

Pathology.—The exact pathological nature of this disease has not been clearly made out, and opinions vary greatly as to its causation and character. It has been suggested that this disease is typhus, to which inflammation of the membranes of the brain and spinal cord is added. There is always some congestion of the brain and spinal cord found after death. It appears to prevail most frequently among soldiers, and, as a rule, during the winter months. Most countries have been visited by the disease, and although certainly infectious, it does not appear to be as markedly so as typhus; in fact, in its mode of propagation it more closely resembles cholera and typhoid than typhus. It attacks all ages indiscriminately, but chiefly the young, from fifteen to thirty years. Cerebro-spinal meningitis is generally fatal, the average number of deaths being about sixty per cent.

Treatment.—So uncertain in their curative effects are the various methods of treatment that have been suggested for cerebro-spinal meningitis, that the practitioner will find it better to rely on general principles, and adopt those measures which the case appears to demand—thus, ice to the head, chloral or bromide of potassium to relieve pain or uneasiness, etc. Purgatives to relieve the bowels if necessary, blisters to the back of the neck, or the application of belladonna to the same part, may be tried. Opium and quinine in large doses, frequently repeated, have found favour in America. The free administration of stimulants may be needed.

YELLOW FEVER.

Lat. *Febris flava.* Fr. *Fièvre jaune.* Ger. *Gelbes Fieber.* Syn. *Gelfieber.*

DEFINITION.—A malignant epidemic fever, usually continued, but sometimes assuming a paroxysmal type, characterised by yellowness of the skin, and accompanied in the severest cases with hæmorrhage from the stomach (black vomit), nares, and mouth.

TYPHUS ICTERODE—VOMITO NEGRO—BULAM FEVER.

Yellow fever is essentially a disease of tropical climates, seldom extending beyond 40° north or 20° south latitude. The exact origin of the poison of yellow fever has not been clearly made out; it would appear, however, not to be due to malaria, but more probably to the accumulation of fæcal excreta round dwellings, and to overcrowding. Parkes says, "During the last few years the progress of inquiry has entirely disconnected true yellow fever from malaria, though yellowness of the skin is a symptom of some malarious fevers. Yellow fever is a disease of cities, and of parts of cities, being often singularly localised like cholera.

In its frequent occurrence in non-malarious places, in the exemption of highly malarious places, in its want of relation to moisture in the atmosphere, and in its evident connection with putrifying fæcal and other animal matters, its cause differs entirely from malaria." A continued high temperature (72° to 77° F.) of some weeks' duration greatly influences its origin and spread. It prevails epidemically chiefly in towns situated on the seashore, and in most cases begins in those parts nearest the harbour. The negro race appear to possess an almost absolute immunity from this fever. The white races, and those recently entering the yellow fever zone, are most susceptible. It is stated by La Roche on the authority of Deveze, that curriers, tanners, soap-boilers, candle-makers, and in general all those who habitually breathe an unwholesome atmosphere, are not liable to the disease. Dr Rush noticed a like immunity among butchers. On the other hand, locksmiths, bakers, and all those who habitually go near furnaces, are readily attacked by the disease *(Deveze)*. Opinions vary greatly as to the contagious or non-contagious character of this fever. Whatever may be the nature of the yellow fever poison, it has, however, been clearly proved to be portable, and that it can be introduced into a town by *fomites*, and this fact alone proves that the cause or causes of yellow fever and paroxysmal fevers are entirely distinct. Improper food, drunkenness, sexual excesses, and neglect of proper hygienic measures are conducive to its origin and spread. The period of incubation varies from two to fourteen days.

Symptoms.—A sensation of chilliness, with or without rigors, followed by supra-orbital headache, pains in the limbs and back, anorexia, constipation, and vomiting of a limpid opalescent matter, together form the symptoms met with in an early stage of yellow fever. Should the disease continue unchecked, the vomit assumes the characteristic black colour, and a fatal termination is to be apprehended. The attack, however, in most cases, comes on suddenly, without any premonitory symptoms. The vomited matter consists of blood changed by the action of the gastric fluids, and varies in colour from a claret or dark-brown to almost black, with a sediment like coffee grounds. The temperature of the body for the first few days rises to about 104–105°, or even 110° F. *(La Roche).* About the fourth or fifth day defervescence sets in, the thermometer marking a steady fall to normal or below this. The pulse seldom exceeds 100, sometimes sinking as low as 40 or 30 beats a minute. Respiration is quick and superficial. The tongue, at first moist and more or less coated, becomes smooth, dry, and red like raw meat as the disease progresses. The urine is always acid in the first stage; during convalescence it is alkaline. Sometimes this secretion is entirely suppressed. The colour for the first few days of the fever is normal, but it soon assumes a sulphur or primrose tint; it then deepens to yellow or orange, and, should the patient recover, may appear dark brown or black. Albuminuria is of frequent occurrence. The skin in the course of the disorder assumes a yellow colour, whence the name. Occasionally coma and convulsions may occur, most probably due to uræmia. Delirium is sometimes present. Five typical forms of this disease have been described:—

1. The algid form.
2. The sthenic form.
3. The hæmorrhagic form.
4. The purpuric form.
5. The typhous form.

The *post-mortem* examination reveals no special anatomical characters. The liver appears to be most frequently affected. The colour is unnaturally yellow in some cases, in others a state of fatty degeneration is perceptible. On the œsophagus and stomach may sometimes be found ecchymoses and erosions. Louis says:—"The lesions which we have placed before the reader were rarely considerable, very often insufficient to explain the death."

Diagnosis and Prognosis.—Yellow fever is distinguished from remittent fever by the absence of any affection of the spleen in the former disease, and also by the efficacy of quinine in the latter—yellow fever is contagious, remittent fever is not; and from small-pox by the presence of the peculiar eruption of that disease. From relapsing fever the diagnosis is made by taking into consideration the etiology of the two diseases. A severe form of relapsing fever with jaundice that once occurred in Dublin was unrecognised, and described as "British yellow fever." The prognosis in a case of yellow fever should always be very guarded; the presence of black vomit is of evil omen. The absence or gradual disappearance of albuminuria is a favourable sign.

Treatment.—With regard to the treatment to be adopted, most observers agree that in mild cases the less interference the better. Purgation is contra-indicated except in those cases which call for depletion, or when uræmic poisoning is feared. The gastric irritation must be treated on general principles—creasote, small doses of prussic acid, or ice to allay the sickness. Mustard plasters, or small blisters applied over the pit of the stomach, will often afford relief. Hot baths and warm pediluvia are of great service. As a prophylactic quinine is useless. On the whole then, careful hygienic conditions, the use of supporting measures when necessary, the administration of diaphoretics and the chlorate of potash, will afford the greatest hope of success in the treatment of yellow fever. The following measures should be adopted immediately the disease shows itself in a barrack or town:—

1. Complete isolation of those attacked. Evacuate the barracks.
2. Perfect sewerage and ventilation.
3. Good water, freed from any possibility of contamination.
4. Patients are best treated in the open air if the season of the year permit.

5. Remove all discharges at once, and mix them with sulphate and chloride of zinc. In ships the bilge-water should be pumped out, and the vessel fumigated. With regard to disinfection, fumigations with nitrous acid gas are said to be the only successful means at present known.

6. Rigid quarantine. It must be borne in mind that in this disease, as in many others, persons not affected may convey the fever to others. It is not necessary that those conveying the disease be themselves affected.

ERUPTIVE FEVERS.

The diseases now to be considered and classed under the head of Exanthemata, or Eruptive Fevers, each arise from a specific contagion, run a regular definite course, and are accompanied with a specific inflammation of the skin, called "the eruption," which also runs a definite course. The mucous membranes are likewise more or less affected.

The members of this group occur for the most part but once during life. The true exanthemata having all these characters are—Small-Pox, Measles, and Scarlet Fever; there are many less perfect, as Chicken-Pox, and the Continued Fevers, but in the latter the eruption is less constant and less prominent than in the true Eruptive Fevers. By some, these diseases have been classed under the head of Zymotic, from the theory that the poison acts on the blood like a ferment. There are five well-marked stages in the career of these fevers :—

1. A period of incubation.
2. A stage of invasion.
3. A stage of eruption.
4. A stage of desiccation.
5. A stage of convalescence.

A TABLE SHOWING POINTS OF DISTINCTION BETWEEN SMALL-POX, MEASLES, SCARLET FEVER, AND CHICKEN-POX.

	SMALL-POX.	MEASLES.	SCARLET FEVER.	CHICKEN-POX.
Period of Incubation....	12 days.	10 to 14 days	4 to 6 days.	Uncertain.
Premonitory Symptoms...	Pains in the limbs and especially in the *back.* Sudden chills and rigors.	Those of a catarrhal attack.	Chills, rigors, and sore throat, intense heat of skin.	Slight feverishness and malaise.
Eruption appears........	Third day of fever as a papule with a shotty feel, then vesicular, and lastly pustular. First on face, especially on forehead, back of the wrists, and then spreads over body.	Fourth day of fever. First on face close to the hair, and then spreads over body in crescentric patches.	Second day of fever. First on neck and upper part of chest and inner sides of the thighs, then spreads over face and body.	In some cases after two or three days' fever. First on shoulders and chest. Few spots on face. Most frequently there is no preliminary fever.
Eruption fades..........	Scabs form on ninth or tenth day of fever, and fall off about the fourteenth.	Seventh day of fever.	Fifth day of fever.	The contents of the vesicles become cloudy on the second day, and dry upon the fourth.

Table—*Continued.*

	SMALL-POX.	MEASLES.	SCARLET FEVER.	CHICKEN-POX.
Character of the Eruption.	At first a distinct papule, elevated, acuminated, of a florid red colour, and giving a peculiar "shotty" feel to the finger. The papules become vesicular and then pustular, and lastly a scab is formed.	Papules of a dingy red colour, but slightly elevated, and having a tendency to form crescentric patches. To the finger they give the sensation of being passed over a well-worn nutmeg-grater.	A crimson rash composed of minute, slightly elevated dots. The rash extends over the whole of the body, which has the appearance of a boiled lobster. Desquamation follows.	Small red spots which in a few hours become limpid vesicles about the size of a pea. The surface of the body looks as if sprinkled with water. The contents of the vesicles become cloudy but never purulent. The vesicles dry up and the crusts fall off, seldom leaving any cicatrix.

VARIOLA—SMALL-POX.

Lat. *Variola.* Fr. *Variole.* Ger. *Blattern.*
Syn. *Menschenpocken.*

Definition.—A contagious and infectious continued fever, resulting from the absorption of a specific poison, and characterised by the appearance of a peculiar eruption, commencing on the third day of the fever.

Small-pox has been divided into two varieties—*variola discreta* and *variola confluens.* In the first variety all the pustules are *separate* and *distinct;* in the second they coalesce over the greater part of the body. There are also varieties known as *corymbose,* when the eruption is arranged in clusters; *malignant; hæmorrhagic; benigna,* when the pocks dry up without becoming purulent; and *variola sine eruptione.*

Confluent small-pox has a tendency to become malignant, a form of the disease which, when once seen, is not likely to be forgotten. A case once occurred in the practice of the writer in the person of a pot-boy engaged at a small public-house in London. The patient, who was suffering from the disease in a mild form, left his bed to proceed to the house where he was employed to transact some matters of business, utterly regardless of the consequences to himself and others. On his return he was caught in a shower of rain, and he reached his home wet to the skin. I saw him the next day, when I found him very ill; on the next day when I visited him, his face, hands, and lips were much swollen, and the whole surface of his body was a light-mahogany colour. On the following morning I found him dead, the colour of the body being now of a dark mahogany. I was informed that during the night he had been delirious, and had attempted to throw himself out of the window, having been restrained with difficulty from doing so; that towards morning he had become more composed, and had only been left for a few minutes when a sudden fall on the floor had brought his attendants back, to find him dead. He had apparently got out of bed to pass water, and had died in the attempt.

There are four well-marked stages—

1. A period of incubation of from twelve to fourteen days' duration.

2. A stage of invasion.	Two to four days.
3. A stage of eruption.	(*a*) Eruption about five days. (*b*) Suppuration four or five days.
4. A stage of desiccation.	Six to ten days.

Symptoms.—After a period of incubation an attack of small-pox is generally ushered in by a sensation of chilliness, alternating with flushes of heat. Nausea followed by vomiting is often an early symptom. The tongue becomes furred, the throat sore, and pain is complained of in the epigastric region. Cephalalgia, pains in the limbs, *back*, and loins are also present. At this stage the disease may be mistaken for a severe bilious and catarrhal attack. Pain in the loins is an important diagnostic symptom, and so also are the frequent perspirations. Children are often seized with convulsions in this early stage. On the second day of the disease the thermometer may mark 105° F. of heat *(primary fever)*; the temperature then falls on the appearance of the eruption, but during the period of suppuration *(secondary fever)* it again rises to 102° or 105° F., the evening temperature being somewhat higher than the morning. This irregular course of rise and fall continues till about the eighteenth day, when the normal temperature is reached. A peculiar disagreeable smell rises from the patient during maturation and bursting of the pustules. In the confluent form of the disease, to the above is added greater severity; the pain in the loins is greater; the tonsils and parotid glands are inflamed and swollen; the eyelids swollen, and the eyes closed; the lips are puffy, and a constant flow of saliva dribbles from the corners of the mouth; the urine becomes scanty, but never suppressed, and charged with urea and uric acid; albumen for a few days is sometimes present, and even blood in severe cases may appear in the urine. The pulse is feeble and quick, and symptoms of increasing debility make their appearance. The secondary fever, usually slight in the distinct variety, becomes intensified in the confluent, and the patient may die suddenly, unable to contend against the virulence of the attack. Delirium in these cases is of frequent occurrence, and the danger to life becomes great, the most fatal period being between the eighth and thirteenth days, but especially the eleventh.

Description of the Eruption.—The eruption runs a definite course, appearing first on the forehead, neck, and backs of the wrists in small isolated inflamed points, called *papulæ;* these, extending thence over the body and lower extremities, on the fifth day become *vesicular*, the cuticle being raised and filled with serum. On the seventh day each vesicle becomes umbilicated by the appearance of a central depression. On the eighth day the vesicles suppurate and become *pustular*, the central depression is lost, so that the pustules are orbicular, and are surrounded by a bluish inflamed ring, termed an *areola.* About the eleventh day the process of scabbing takes place, the pustules bursting, and then gradually drying up. The fever, as soon as the eruption appears, *remits*, but returns during the suppurative stage of the pustules. This return of the fever *(secondary fever)* is considered by some to be due to the specific nature of the disease, by others to result from the process of maturation in the pustules, and to partake somewhat of the nature of a *suppurative* fever, *symptomatic* of the local affection.

Complications, etc.—Beside the secondary effects of the small-pox poison on the system—the fever and the eruption—there are well-marked *tertiary affections*, which, for the sake of convenience of description, we shall tabulate as follow :—

(*a*) Vascular System.
(*b*) Genito-urinary System.
(*c*) Cutaneous System.
(*d*) Digestive System.

(*a*) *Vascular System.*—Hæmoptysis is by no means of unfrequent occurrence, and this may or may not be combined with inflammation of the lungs, which generally takes the form of pleuro-pneumonia. When the latter occurs, the mucous membrane of the trachea is often found covered with an irregular honeycombed layer of semi-purulent, muciform matter, which is said to be peculiar to small-pox. This on removal leaves the subjacent tissue in a state of diffuse inflammation. Dr Gregory asserts that the pleura is particularly liable to take on an inflammatory action, and that this generally occurs about the eleventh or twelfth day. Well-marked pyæmia may also occur when the disease becomes confluent, and in one case, where this condition was present, the increase in the sulphuric acid passed in the urine was well marked. The muscular structure of the heart is also often softened and easily penetrated by the finger.

(*b*) *Genito-urinary System.*—Hæmaturia, renal abscess, and orchitis are the most frequent complications in the male; whilst in the female, ovaritis, menorrhagia, and abortion, should the woman be pregnant, together with a like tendency to hæmaturia and abscess of the kidney.

(*c*) *Cutaneous System.*—The areolar tissue under the skin is in some cases the seat of numerous small abscesses, which form in rapid succession on the limbs. These abscesses seldom contain healthy pus.

The cuticle is frequently softened, and has a marked tendency to peel off.

In confluent small-pox the vesicles on the body and extremities, though often not confluent, are pale, and have no areola, and the face is pale and doughy. The vesicles have by careful dissection been dissected out, and it has been found possible to puncture the vesicular portion of the eruption and let out its contents without disturbing the pustular portion, which may be also treated in a like manner; the eruption, in fact, consisting in a vesicle surrounded by pus, placed in a cup-shaped cavity. The pitting of small-pox is the result of the extension of the pustular affection into the true skin, or *rete mucosum*, and the destruction of the part thus attacked.

(*d*) *Digestive System.*—Whether the pustular eruption of small-pox is ever present on the mucous membrane of the stomach and intestines may admit of doubt. It may, however, occur in the mouth and on the fauces, giving rise to the sore throat so often complained of by the patient. The probable explanation of their supposed presence on the intestinal mucous membrane, may be that suggested by Aitken, who remarks that "the appearance of eruption in such cases is due to the solitary mucous glands, which are filled with exudation, not of a purlent kind, but having all the external appearance of pustules."

The *sequelæ* are, deafness (due to suppuration of the internal ear), blindness, abscesses, boils, etc.

With regard to the affection of the eye, often ending in blindness, the late Mr Marson says:—"The eye seems to possess a complete immunity from the small-pox eruption, and that although it sometimes extends to the inner margins of the eyelids, the particular local affection that causes the destruction of the organ of vision in variola begins generally on the eleventh or twelfth day, or later, from the first appearance of the eruption, and when the pustules in every other part of the body are subsiding. It comes on after the secondary fever has commenced, with redness and slight pain in the part affected, and very soon an ulcer is formed, having its seat almost invariably at the margin of the cornea. This continues to spread with more or less rapidity, and the ulceration passes through the different layers of the cornea, until the aqueous humour escapes, or till the iris protrudes. In the worst cases there is usually hypopion, and when the matter is discharged, the crystalline lens and vitreous humour escape. In some instances the ulceration proceeds very rapidly. I have more than once seen the entire cornea swept away within forty-eight hours from the apparent commencement of the ulceration; and, what is singular, now and then the mischief goes on without the least pain to the patient, or his being aware that anything is amiss with his eyes."

Diagnosis and Prognosis.—The diagnosis prior to the stage of eruption cannot be made with certainty. In measles, the stage of invasion is longer, and is marked by catarrhal symptoms; in scarlet fever, the rash appears on the second day, and the throat is early affected (see Table, p. 68). Small-pox, as a rule, occurs but once in the same person, but cases are on record of two, three, or even more attacks in the same individual. It may also be combined with measles, whooping-cough, ague, etc. etc. In these cases, it is more or less modified by the concurrent disease.

The fatality in natural or unmodified small-pox is great, but the danger to life is greater in some epidemics than in others. The confluent variety is more fatal than the distinct, the danger in the former being greater during the stage of the secondary fever. The eleventh day of the fever, or the eighth day of the eruption, is the period at which deaths most frequently occur. Death is generally due to a combination of asthenia and coma. The danger to life is greater among children than adults. Pregnant women are said, as a rule, to abort and die. I once attended a woman five months gone in pregnancy. She did not abort, made a good recovery although she had the disease very severely, and her child, when vaccinated, presented an excellent arm.

Dr Watson says:—"There is no contagion so strong and sure as that of small-pox: none that operates at so great a distance." Unequivocal evidence of minute scales of small-pox matter have been detected by Bakewell in small-pox wards, and these may be the means of propagating the disease. Small-pox is more contagious in its later than earlier stages.

Treatment.—The treatment of small-pox is either local, pertaining to the eruption, or general. The general treatment, when the disease is distinct and slight, consists in placing the patient in a large, well-ventilated room, or better still, in a tent or wooden shed. A brisk saline purge may be administered at the outset, and a mixture containing the chlorate or citrate of potash may be given, but as a rule the less medicine the better. Treat complications as they appear; otherwise, there is no special treatment. Barley-water, lemonade, and iced drinks may be allowed, if there be great thirst. In confluent small-pox, the treatment must be varied to meet the symptoms as they arise. Opiates are indicated if there be great restlessness and delirium. If the pulse become feeble and the patient exhausted, supporting measures will be required; strong beef-tea, tincture of bark or quinine, and alcohol, if necessary, freely administered. In the hope of preventing the spread of the disease, the patient may be smeared with oil and a little carbolic acid, or glycerine and carbolic acid. The permanganate of potash in solution, or diluted sulphurous acid may be used as washes for the mouth. The clothes should be baked, if possible, before washing. All the attendants on small-pox cases should either have had the disease or been recently vaccinated.

The following are some of the suggestions that have been offered with regard to the local or *ectrotic* treatment of the eruption:—

(*a*) Evacuation and cauterisation of the pustules with nitrate of silver.

(*b*) Exclusion of light, placing the patient in total darkness. Some suggest yellow light.

(*c*) Opening the pustules with a fine needle.

(*d*) Smearing the skin with mercurial ointment.

(*e*) Painting the surface with collodion.

Many other measures have been proposed at various times to obviate the disfigurement caused by *pitting*, but these efforts have not hitherto met with any decided success.

VARIOLOID.

Under this head are now classed such diseases as were formerly known and described as *stone-pock*, *horn-pock*, *wart-pock*, but which are now considered to be small-pox more or less modified by vaccination.

The following list of differences between small-pox and varioloid, given by Aitken, will help to distinguish the two diseases:—

1. A fever of three days, without eruption, affecting people during variolous epidemics.
2. A high and severe fever, followed by a mild eruption, sometimes only a single pock; the slight proportion which the amount of eruption bears to the severity of the preceding fever is, perhaps, the most marked characteristic of varioloid.
3. The occasional appearance of a scarlet efflorescence, like that of a scarlatina or roseola, preceding the appearance of the proper pimples, which occur as a scanty crop.

4. In some rare instances the eruption is confluent, but does not advance beyond the development of a pimple or vesicle, and begins to dry on the fourth or fifth day of the eruption, forming a small hard tubercle, which soon disappears.
5. Sometimes the eruption is at once pimple, vesicle, and pustule at one time in the same case.
6. Sometimes the eruption runs its regular course, but stops sooner, sometimes on the sixth or seventh day, instead of the eight or ninth. In general, it may be stated that the severity and fully developed state of the disease is in proportion to the length of time which elapses from vaccination.
7. The varioloid eruption wants the peculiar odour of natural small-pox, and secondary fever is very rare.
8. Other eruptive affections—such as measles, scarlatina, purpura—materially modify the course and symptoms of small-pox.

Treatment.—As in mild forms of small-pox.

VACCINIA.—COW-POX.

Lat. *Vaccinia.* Fr. *Vaccine.* Ger. *Kuhpocken.*

Definition.—A contagious disease transmitted from the cow to man by vaccination or inoculation.

To Dr Jenner is due the discovery, that cow-pox communicated to man renders him less susceptible to the contagion of small-pox. The generally received, and probably the only correct, opinion is, that vaccinia, or cow-pox, and variola are identical, the poison of the latter being modified, and rendered milder by passing through the system of the cow. This view has, however, been of late attacked by a few who maintain that small-pox is entirely distinct from cow-pox; that small-pox is with difficulty produced in the cow, and that when the operation is successful, the lymph produces veritable small-pox when introduced into the human subject.

There are several methods by which the operation of *vaccination* may be performed, but the following may be taken as a guide. The usual place chosen for implanting the lymph of cow-pox is the skin covering the deltoid muscle of the arm. The arm is grasped by the left hand of the operator, and the skin being tightened, a clean sharp lancet, charged with lymph, is passed gently into the skin, so that a valvular puncture is made, and the vaccine virus allowed to enter the wound.

If the operation be successfully performed, about the second day small red points appear raised over the sites of the punctures. Papules on the fourth day become developed, and on the fifth or sixth day distinct vesicles are discoverable. On the eighth day they attain to their full development, and are surrounded by an areola. On this day also the vesicle is distended by a clear lymph. About this time there is usually some slight fever, the lymphatics of the arm are swollen, and the axillary glands enlarged. Soon after the eighth day the contents of the vesicles become more or less purulent, and a black spot is seen in the centre of the pock. On the fifteenth day a hard blackish scab is formed, which generally falls off about the twenty-fifth day from the date of the operation. A lichenous eruption often attends or follows vaccination. The best age at which to perform the operation is before the child is three months old, and before the process of dentition has set in. The child should also be in the enjoyment of sound health.

Saline aperients and the application of a cold bread-and-water poultice to the inflamed pocks, is all that is necessary by way of treatment.

VARIGELLA, OR CHICKEN-POX.

Lat. *Varicella.* Fr. *Varicelle.* Ger. *Windpocken.*
Syn. *Wasserpocken, Varicellen.*

DEFINITION.—A contagious disease, occurring chiefly in early life, attended with a vesicular eruption and slight fever.

Chicken-pox requires no special treatment beyond the restriction of the diet and the administration of saline aperients. The vesicles usually dry up between the fourth and seventh day of the disorder, leaving small red spots, but seldom cicatrices on the skin. It is of importance to early distinguish this disease from variola, so as to allay the anxiety of the patient's friends.

The following description may help the student to distinguish small-pox from chicken-pox :—

In small-pox the premonitory symptoms are well marked—rigors, considerable fever, and severe pain in the loins.

In chicken-pox, on the other hand, the fever, if present, is slight, and the patient—most frequently a child—scarcely complains of feeling ill; "the body, in varicella, has the appearance of having been exposed to a momentary shower of boiling water, each drop of which had occasioned a minute blister." The vesicles do not become pustular unless irritated.

In some cases the disease is so severe that it cannot easily be diagnosed from small-pox. The diagnosis will be assisted by noticing that the severe symptoms seldom last twelve hours; thus the child will appear very ill at night, and almost well the next morning. The eruption may also be confluent in some cases. The rapidity of recovery, the absence of pitting and freedom from the complications attendant on small-pox, will help the diagnosis.

Treatment.—Simple saline febrifuge medicines are alone required.

SCARLATINA, OR SCARLET FEVER.

Lat. *Febris rubra.* Fr. *Scarlatine.* Ger. *Scharlachfieber.*

DEFINITION.—A contagious and infectious fever, characterised by a high temperature; sore throat and a scarlet eruption appearing on the second day of the fever.

There are three varieties of scarlet fever :—

SCARLATINA SIMPLEX. *Definition.*—A scarlet rash, with redness of the throat, but without ulceration.

SCARLATINA ANGINOSA. *Definition.*—A more severe form of the disease, with redness and ulceration of the throat, and a tendency to the formation of abscess in the neck.

SCARLATINA MALIGNA. *Definition.*—The throat tends to slough; the scarlet rash is scarcely, if at all, visible, petechiæ are often seen on the surface, and the fever is of a low form.

Symptoms.—After a period of incubation of from four to six days' duration, scarlet fever sets in with a sensation of chilliness, rigors, vomiting, and *sore throat*, the last being an early and a prominent symptom. The temperature of the surface rapidly and steadily rises, and attains a greater elevation in this than in the other eruptive fevers, and, unlike small-pox, does not abate at the appearance of the eruption. If the throat be early examined, redness of the fauces, more or less vivid, may be observed, and there is some pain experienced during an attempt to swallow. Hæmorrhage from the nose in this stage is not uncommon.

This is the stage of invasion, the scarlet eruption appearing on the second day of the fever. The eruption is generally first noticed on the body and limbs before it makes its appearance on the face. It occurs primarily in the form of small red dots, more or less raised above the surface of the skin. These dots soon coalesce, forming irregular patches, varying in size and shape. The redness is scarlet, of a boiled lobster colour as described by Watson, and is brightest at the bends of the joints, *inner parts of the thigh*, and the *lower part of the abdomen;* but is not so uniform as in the case of erysipelas, and disappears temporarily on pressure—the reappearance being from without inwards, that is, towards the point where direct pressure was made. The maximum of diffusion and intensity of the rash is reached on the third day after its first appearance, after that period it begins to fade. In some cases, more frequently in the adult than in the child, the rash is altogether wanting, the only signs of the disease present being the continued high fever and sore throat. These cases are not unfrequently followed by albuminuria, leading one to refer the previous suspicious attack to its right source—scarlet fever. I remember a case occurring just before my student's days, where the child of a connection of mine suffered from slight feverish symptoms and a rash, which was pronounced to be merely due to disordered stomach. Some few days after, his cousin, living in the same house, a few years his junior, sickened and died of scarlet fever in its most malignant form. I mention this case to put the student on his guard, and I would warn him to be very careful in his diagnosis.

In scarlet fever, the temperature on the evening of the second day is about 105·6° F., it may then rise on the third day to 105·8° F., and from that day till about the ninth day it vacillates between 103·8° F. and 102·9° F. On the tenth day it falls to 100·9° F., and then defervescence continues uninterruptedly till the normal temperature is reached about the fifteenth day. Dr Sydney Ringer has shown that the temperature in scarlet fever falls on the fifth day of the disease, then on the tenth, and so on, each fall being separated by an interval of five days. Compare this course in the temperature of this disease with that of measles; the sudden fall from 109° F. on the fifth day to 102° F. on the seventh day is typical of the latter disease.

The throat in mild cases of scarlet fever is not very sore, some patients scarcely complaining of any inconvenience. In *scarlatina anginosa* and *maligna*, on the other hand, the affection of the throat is always a prominent and distressing symptom. The glands of the neck become secondarily affected, inflamed, and painful. The appearance of the tongue is peculiar; at first it is greatly furred; the papillæ, projecting through the coating, give it the appearance of having been sprinkled with red sand. As the disease progresses the coating peels off, leaving the surface of the tongue clean and red, the enlarged papillæ giving to it a *strawberry-like* appearance, especially at the tip and edges.

During the eruptive stage delirium generally exists, often requiring the attendance of a watcher to prevent the patient from injuring himself. Slight albuminuria is often present. The duration of the stage of eruption is from four to six days.

Desquamation usually sets in prior to the entire disappearance of the rash. At first the cuticule comes off in small scales like scurf, but it can at times be removed in small sheets, especially from the hands and feet. Sometimes even the nails are exfoliated, and even when this does not occur, transverse grooves may often be seen on them, showing an arrest of their growth and pointing to an explanation for the origin of sequelæ, which otherwise would have remained unexplained These grooves in time disappear, and although they are not peculiar to scarlet fever, their presence is, to say the least, suspicious. Troublesome pruritus, often intense, accompanies this stage. Albuminuria is also present, but, unless in a very large amount, does not indicate any alarming complication.

Not unfrequently, during the course of scarlet fever, the patient complains of severe pains in the joints of a rheumatic character, which often prevent his sleeping.

So far, we have only described a mild case of scarlet fever; but others, unfortunately, too frequently occur when the disease assumes a most malignant type. In *scarlatina anginosa* and *maligna* the whole force of the poison seems to be directed to the throat and cervical glands—the latter becoming so enlarged as to form a great lump on both sides of the neck. The tonsils are enlarged so as almost to block up the entrance to the pharynx, and are covered by a diptheritic exudation. The act of deglutition is performed with difficulty, often with great suffering, and the breath becomes fetid. The rash is slight, sometimes absent. The severity of the throat affection often leads to a rapidly fatal termination, death, in some cases recorded by Dr Kennedy of Dublin, taking place in two days from the commencement of the disease. Towards the fatal termination diarrhœa may set in, or the child may appear to have taken a turn for the better, swallowed some milk or beef-tea with eagerness, and then died in the course of three or four hours. This eagerness for food on the part of patients suffering from the worst forms of this disease, and in fact not in this disease alone, I have repeatedly found, is a sure sign of approaching death.

The *sequelæ* of scarlet fever are albuminuria and dropsy, generally coming on during the stage of desquamation, if care has not been taken to protect the patient from cold. I have seen an attack of dropsy, the first intimation that a patient had been through a mild and unrecognised attack of scarlet fever, while subsequent cases of scarlet fever in the same house more than verified the previous suspicions. Deafness is not an unfrequent sequel, and may be due to perforation of the membrana tympani, to closure of the eustachian tubes, or to disorganisation of the internal ear by the extension of the inflammation from the throat.

When dropsy occurs, œdema is generally first noticed in the face and lower extremities.

Convulsions and coma, due to uræmic poisoning, may also occur as sequels to scarlatina.

Diagnosis and Prognosis.—The early diagnosis of scarlet fever is by no means easy, and the student will often be sorely troubled to decide as to the nature of the case before him. During an epidemic of any of the eruptive fevers, we not unfrequently meet with cases presenting symptoms more or less identical with those of the prevailing epidemic, which, however, pass over, and prove by their termination to be perfectly harmless. During the epidemic of small-pox (1876–1877), many cases of what appeared to be at first sight scarlet fever occurred. There was present slight fever, sore throat, and an eruption closely resembling that of scarlet fever. All these symptoms passed off in a few days, and the patients, mostly children, appeared in their usual health. These cases may be, as some suppose, mild forms of scarlet fever; at any rate, they greatly embarrass the medical man. I have myself seen persons walking about with the small-pox, apparently unconscious of the nature of their illness; and that these cases were in reality small-pox, was rendered beyond suspicion by the fact that they occurred in houses in which a severe case of that disease was under treatment. From what has been before said, however, the practitioner will, we hope, be on his guard, and be careful before he offers too hasty an opinion, and he had better err on the side of too much caution than fall into the mistake of over-precipitation in pronouncing the disease harmless, and thus risking the lives of other members of the family to whose assistance he has been summoned.

The *Prognosis* must be guided by the nature of the symptoms and the severity of the throat affection. Convulsions and coma are often the forerunners of a fatal termination. Death may sometimes be due to the formation of a clot in the right cavities of the heart.

Scarlet fever is very infectious, and may be communicated by fomites. In spite of the greatest care to properly cleanse and disinfect houses, scarlet fever may again break out in the same building after the lapse of some months. One attack is generally a preventive to a second, but the exceptions to this rule are by no means rare.

Among the infectious diseases scarlet fever ranks second in its fatality, being, however, more fatal in towns than in the country.

Treatment.—In simple cases, little is necessary by way of treatment beyond the usual measures adopted in fevers, viz., good ventilation, and the prevention of exposure to cold. Cooling drinks to allay thirst, and simple nourishment may be given. The bowels should be relieved if necessary by the administration of enemas or saline aperients.

There is no specific for the cure of scarlet fever; sound common sense, united with careful watching of the case, so as to be ready to meet every untoward symptom as it arises, is superior to all reputed specifics. The affection of the throat is best treated by a gargle of the chlorate of potash and raspberry-vinegar, or a weak solution of the chlorinated soda. In the case of young children the lozenge of the chlorate of potash (B. P.) may be sucked along with a black currant lozenge; and in this way I have succeeded in disguising the taste of the chlorate of potash, and cheating my young patient. Some recommend the application of the wet sheet, or *wet pack*, as it is called. Sponging the body with tepid water will often afford great relief, and help to diminish the intense heat of the skin. Of course, in the malignant forms of the disease alcohol must be given; bark and the chlorate of potash, or carbonate of ammonia, will form the chief medicines from the administration of which any hope may be derived. The dropsy must be treated by the exhibition of the tincture of iron, and by free purgation with elaterium or jalap. Extreme restlessness and vigilance may require the administration of anodynes; hyoscyamus and belladonna are to be preferred to opium.

During the period of desquamation the patient may be well rubbed with mutton suet or carbolic acid and oil; and then placed in a warm bath, care being taken to prevent cold. Dry cupping behind the loins, or hot fomentations to the same parts, are of great use when the kidneys become affected. Lastly, be careful in your recourse to alcohol, for by beginning its administration too soon, you rob yourself of a valuable assistant in the hour of need.

DENGUE.

Lat. *Denguis.* Fr. *Dengue.* Ger. *Dengue.*

Definition.—To a contagious disease, occurring epidemically, characterised by pyrexia, accompanied with excruciating pains in the head, small joints, muscles of the body and extremities, and with the presence of an eruption at one time like that of scarlet fever, at another like that of measles or urticaria, the name Dengue has been given.

Of this disease little is known, the first mention we have of its appearance being an account of an outbreak which occurred among a body of troops at Rangoon in 1824. It has been called by some *break-bone fever*, or *dandy fever*, and by others *scarlatina rheumatica.* Epidemics have occurred in the West Indies, and in several parts of the United States as described by Rush in 1780.

Symptoms.—Those present in all fevers—hot skin, quick pulse, thirst, etc. The onset of the disease in some cases is insiduous, in others sudden. The most important feature of the disease is the terrible rheumatic-like pains in the joints, which are so distressing to the patient. Relapses, accompanied with a *second* appearance of the rash on the palms of the hands, the feet, and knees are not unfrequent, an attempt at convalescence being suddenly interrupted by a return of all the symptoms. The disease is fortunately seldom fatal except in the case of infants.

Treatment.—The treatment consists in free purgation and in the administration of opiates to relieve the suffering, and the application of rubefacients to the spine and painful parts. Blisters may be of use over the painful joints. Alcohol is often indicated. During convalescence, which is often tedious and prolonged, tonics and change of air will be required.

MORBILLI, RUBEOLA, OR MEASLES.

Lat. *Morbilli.* Fr. *Rougeole.* Ger. *Masern.*

DEFINITION.—A continued fever, commencing with well-marked catarrhal symptoms, the presence of a mulberry-coloured rash, appearing on the fourth day of the fever, and disappearing on the seventh.

Like scarlet fever and small-pox, an attack of measles appears to be due to the absorption of a specific poison into the system. The disease can be communicated by inoculation and by fomites. Children are more frequently attacked than adults; but it has, however, been known to prevail as an epidemic among troops. As a rule it occurs but once during life.

Symptoms.—Measles, unlike the other eruptive fevers, sets in with *well-marked catarrhal symptoms.* The eyes water, the nose runs, and the patient presents all the appearance of one suffering from a severe cold.

The period of incubation of measles is from ten to fourteen days, *the rash appearing on the fourth day of the fever*, and fading away on the seventh.

The eruption first appears on the forehead and temples close to the roots of the hair, extending in a few hours over the body and limbs. It is of a dull-red or raspberry colour, occurring in the form of minute dots not unlike flea-bites, slightly elevated above the surface, and having a tendency to coalesce in crescentic forms. When the disease is very severe the eruption assumes a dark purple colour, and all the symptoms are exaggerated. *Defervescence* takes place on the *disappearance* of the eruption, but not on its *appearance* as in small-pox.

During the course of the disease, a troublesome cough annoys the patient, but this rapidly disappears as the eruption fades; nothing is more constantly present, and with the exception of the eruption, more characteristic The typical range of temperature in measles is as follows:—From 106·3° F. on the evening of the fourth day, to 109° F. on the following evening, and then a rapid fall to 102° F. on the morning of the seventh day, with a slight rise in the evening of the same day. About the tenth day the temperature has reached its normal standard.

Bronchitis, pneumonia, diarrhœa, and in severe cases, gangrene attacking the mouth, vulva, nose, and lungs, may occur during the course of measles. In children, chest complications are most frequently present, and *should be carefully looked for and treated*, for it is well to remember that bronchitis occurring in the course of measles is most insidious, ending sometimes in young children in convulsions and death.

Diagnosis and Prognosis.—From scarlet fever measles may be separated by the marked catarrhal symptoms in the latter, by the scarlet colour of the rash in the former, and the dull-red crescentic patches in the latter, and also by the absence of the peculiar sore throat so characteristic of scarlet fever. Measles must not be mistaken for urticaria or roseola. The absence of catarrhal symptoms in urticaria will mark the distinction.

The prognosis is favourable in simple measles, the danger in the young being due to bronchitis, the formation of a diphtheritic membrane on the fauces or larynx, pneumonia, convulsions, enteritis, and other complications.

I have frequently seen measles followed in children by whooping-cough.

Treatment.—The treatment in mild cases consists partly in palliative, and partly in dietetic and hygienic measures. Rest in bed. Aperients when necessary should be given, and the cough, which is often very distressing to the patient, must be allayed by appropriate medicines. The acetate of ammonia, or nitrous ether may be given in a mixture sweetened with syrup, and flavoured with tincture of orange. The cough is best allayed by the following mixture for a child three years old:—℞ Tinct. Camphoræ Co., ʒi; Tinct. Conii, ʒi; Vin. Ipecac., ʒi; Acidi Hydrocyanici dil., ♏x. Syrupi, ℥i; Aquam, ad ℥iss. Mft. Sumat ʒi quartâquâque horâ. The diet should consist of beef-tea and light puddings and milk. Complications, as they present themselves, must be treated on general principles.

RÖTHELN, RUBEOLA, OR GERMAN MEASLES.

Lat. *Rubeola. Notha.* Fr. *Roséole.* Ger. *Epidemisch Roseola.*

DEFINITION.—"A fever ushered in by coryza, redness, and watering of the eyes, redness and soreness of the throat, pains in the head, back, and limbs, attended on the third or fourth day by sudden and general eruption of a red efflorescence, which terminates about the tenth day by desquamation, the disease presenting the character of measles and scarlatina conjoined."—*(Copeland.)*

The disease known by the above names, now for the first time given as a distinct affection in the nomenclature of diseases issued by the College of Physicians as "epidemic rose rash." The disease occurs most frequently in hot seasons, and more often attacks children than adults.

Symptoms.—Premonitory symptoms are usually slight, and may be absent. There is generally some langour and headache, with occasionally nausea, vomiting, and diarrhœa. Catarrhal symptoms, with a harsh, clanging cough, as in measles, are very frequent. One of the most constant premonitory signs known is sore throat, and cases have occurred in which there was inflammation of the tonsils, velum, and uvula. The duration of the invasion stage varies from a few hours to three, four, or five days, and it is succeeded by the appearance of the characteristic rash. The eruption appears usually about the second day, though it may not be seen until the fourth or fifth. It consists of an efflorescence breaking out in some cases at once all over the body; in others, beginning on the sides of the nose and then spreading over the body, and consists of small red stigmata, which run together to form patches of variable size, with irregular margins, not of so distinctly crescentic a shape as those of measles. The eruption is usually at its height on the second day, rapidly disappearing in three or four days *(Bristowe)*. In colour, the patches have been aptly compared by Paterson to the appearance "produced by a writing quill dipped in red ink and having its point placed on moist white paper." In some cases, the patches may so coalesce as closely to resemble the eruption of scarlatina, but careful examination will always discover one or more distinct patches. The eruption is attended with considerable itching. A rash not unlike that of rötheln is often caused when copaiba or chloral hydrate is taken for some time. While the eruption continues, the other symptoms increase, the sore throat is aggravated, and there may be much hoarseness and loss of voice, with considerable external swelling and tenderness. There is also occasionally much internal tumefaction, with total inability to swallow even the slightest portion of fluid, which generally regurgitates by the nose.

The pulse during this stage is frequent, the skin hot and dry, and the temperature rises to about 101° F. Death may occur during this stage by suffocation from the great mucous secretion in the throat. Vomiting is not uncommon. The eruption stage lasts a variable time — from eight hours to eight or ten days, the average being about four or five days. On the rash disappearing, the third stage, that of desquamation, takes place. This commences at the centre of each eruptive patch, and extends towards the circumference. It is of a branny furfuraceous character, the epidermis never being shed in large scales or pieces, as in scarlatina. This process takes from five to twelve or fifteen days.

Diagnosis.—The only affections with which rötheln can be confounded are measles and scarlatina. From them it differs, as will have been seen, in not being accompanied by so high a temperature, in the appearance of the rash at once on the whole body, in the fact that it propagates itself, and then gives rise to either measles or scarlatina; in the characteristic appearance of the eruption; in the manner of desquamation; in the size and brightness of the eruptive patches increasing with the severity of the attack; and in the tongue being more or less dirty at first, then strawberry-like, and finally smooth. Fagge is inclined to doubt the existence of rötheln as a distinct disease, and suggests that it is possibly an anomalous or ill-developed form of measles, a view supported by the contradictory descriptions given by different observers who have written on subject. With this view I am inclined to concur.

An attack of rötheln affords no protection against either measles or scarlatina, and *vice versa*, neither measles nor scarlatina protect against rötheln.

Treatment.—In most cases little treatment is required, confinement to bed, an aperient, and simple saline mixture, being usually all that is required. The throat symptoms may require attention, and for their relief, steaming with hot water, and a gargle of milk and water, may be employed.

PLAGUE.

Lat. *Pestilentia.* Fr. *Peste.* Ger. *Pest.*

Definition.—A specific, highly infectious and contagious fever, attended with bubo of the inguinal or other glands, and occasionally with carbuncles.

The plague is a contagious malignant disease, attended with pyrexia and the presence of buboes, carbuncles, pustules, and petechiæ in various parts of the body. It is now happily confined to Eastern countries, the last visitation in Western Europe being in the year 1665, and known as the "Great Plague." It is computed that no less than 68,526 persons died in one year in London and its suburbs alone. The disease is generally preceded by great languor, headache, shivering, and vomiting. The local phenomena then appear: swelling of the tongue, excruciating pains in the axillæ, and the formation of buboes and carbuncles. Petechiæ soon make their appearance, and spread over the body, and are considered of unfavourable omen. After death, the internal organs are found engorged with blood, the liver is enlarged, and sometimes covered with petechial spots. The kidneys are surrounded by a hæmorrhagic effusion, and are themselves loaded with blood. The disease is to some extent endemic in Egypt.

The treatment of plague is not very satisfactory; but the measures which appear to promise most success are those adopted in the treatment of malignant typhus fever.

EPIDEMIC CHOLERA.

Lat. *Cholera pestifera.* Fr. *Choléra Asiatique.*
Ger. *Cholera; Asiatische Cholera.*

Definition.—An epidemic disease, characterised by vomiting and purging, with evacuations like rice-water, accompanied with cramps, and resulting in suppression of urine and collapse.

Cholera is an epidemic disease due to the admission of a specific poison into the blood, possibly of a miasmatic origin. The exact mode of origin and propagation of cholera has not yet been clearly made out, but it is generally supposed to be due to the reception of a peculiar poison, which rapidly undergoes multiplication within the body. By some authorities the existence of a cholera germ, which, however, has not yet been demonstrated, is believed to be necessary to produce the disease, and some evidence in support of the parasitic view is afforded by the sudden appearance in mid-ocean of cholera on ship-board. The reader is referred to the investigations of Koch and others with regard to the so-called "comma bacillus" of cholera. They are said to abound in the alvine secretions during the height of the disease, but nearly disappear while the secretions contain fœcal matter. No bacilli were found in the water used by the affected, however foul. The probability is that two factors are required—a germ and a peculiar condition of the system. The germ may, by appropriating certain portions of a substance harmless in one condition, and normally existing in the body, so break up the constitution of the substance as to render it poisonous and capable of producing disease. Poisonous substitution products may thus be formed from previously existing harmless ones. Pettenkofer and other observers have shown that in the origin and spread of cholera three factors are necessary—place, time, and individual,—these being usually spoken of as the local, temporal, and individual disposition. We have already, under Typhoid Fever, given Pettenkofer's views as to the relationship between the origin and spread of that disease and the water in the soil, or "ground-water," as he calls it. With regard to cholera, M. Pettenkofer entertains like views; but these, though supported by many ingenious arguments, appear to be negatived by experiences in India. Be this as it may, it appears not improbable, as suggested by the authority just mentioned, that it is the fall and not the accumulated ground-water which is most liable to diffuse the germs of infectious diseases. Cholera is undoubtedly disseminated by means of the discharges from the infected impregnating the water of rivers and wells used for domestic purposes. The well-known history of the Broad Street pump need not be recorded here, but there cannot be a doubt but that the closing of the well stopped the local epidemic of cholera. The atmosphere is also probably more or less influential in its spread; and this is especially the case when the air is surcharged with moisture, as in India during the monsoons. Excessive dryness or wetness of the soil seems to be unfavourable to the spread of cholera. It may be imported also from one locality to another by human intercourse, and by fomites. Lebert doubts the influence of dead bodies as a means of infection.

History.—Although cholera has probably recurred at varying intervals in India from the earliest times, yet from Jessora, in 1817, must we date the origin of those fearful epidemics which from time to time have overrun Europe. Beginning, then, in Jessora in 1817, it spread over the greater part of British India, and from that year it hovered about India, gradually extending its boundaries till 1819, when it passed from the mainland to Penang, Ceylon, Sumatra, the Isle of France, and the Isle of Bourbon. In 1820, after again severely attacking Bengal, it reached the Philippines, entered China, visiting several of the cities of that country. In 1821 it crossed the Indus and ravaged the coasts of the Persian Gulf, and then advanced inwards to Bagdad and Ispahan. In 1822 India again suffered, and the disease now advanced towards Europe by way of Syria and Erzeroum. In 1823 Burmah and China were again attacked, and for the first time it appeared on Russian soil, and visited Orenburg. For about three years cholera seemed to be more or less quiescent, but in 1827 it again appeared in Calcutta, and from that year to 1829 visited Lahore, Cabul, and Orenburg. Sebastopol, Odessa, and Moscow were severely attacked in 1830. In 1831 several Continental cities were visited, including Berlin. From Hamburg it reached England, first appearing in Sunderland on 26th October 1831. In 1832 London was visited in January, Edinburgh in February, and Dublin in March. From England it spread to France, and then, leaping the Atlantic Ocean, appeared in Canada, ravaging Quebec and Montreal. It then entered the United States, proceeding southwards to Mexico. From America it returned to Europe, landing on the coast of Portugal; and after visiting all the countries of Europe, gradually died out in the year 1837 as far as Europe was concerned. After remaining more or less active in India, it again passed into Europe in 1848–49 and in 1855 and in 1866. Brazil was first visited in 1854. With regard to the spread of cholera, an interesting fact has been noticed—that although in its advance towards and through Europe the places attacked were ranged along the great traffic lines, still it was noticeable that, whilst Paris and Marseilles were almost depopulated, Lyons escaped. But, further than this, one part of a town suffered more than another. Munich, Berg, and Flaidhausen were examples of this. In these towns it was almost universally noticed that the houses on the limestone gravel were those attacked, whilst those on the brick clay almost entirely escaped. This singular fact may be explained by the amount of ground air contained in loose, moist soils not saturated with water, and which is hourly drawn into the houses whenever they are warmer than the surrounding atmosphere. In clay soils there is but little of this ground air, and hence the comparative freedom from diseases which it affords. Cholera is generally held to be independent of climatic influences, but it would appear that, taking a number of epidemics that have occurred in different countries, in half the disease was more prevalent in summer than in spring and autumn, and least in winter. In 1830 cholera prevailed in Moscow with a temperature of –4° F. This unusual occurrence might have been due to the fact that the cholera discharges were in many cases thrown on the snow round the houses, which snow was not infrequently melted and the water used for domestic purposes.

Symptoms.—The period of incubation appears to vary from twelve hours to two or three days. In *some* cases an attack of cholera is preceded by simple diarrhœa and vomiting. After this condition has continued for a period more or less prolonged, the specific symptoms of the disease make their appearance. Of the cases of cholera treated in St Bartholomew's during the epidemic of 1866, premonitory symptoms were absent in 43·1 per cent. of fatal cases, and in 21·6 per cent. of non-fatal cases. "My experience," says Dr Church, "would certainly lead me to say that premonitory symptoms are less frequently present in the severer than in the milder cases." In *other cases*, however, the attack comes on suddenly, and rapidly passes into the algide form, death occurring, according to Dr Gavin Milroy, "within little more than five minutes" in hale and hearty men. In cases not so rapidly fatal as those just mentioned, the premonitory symptoms as above mentioned are simple diarrhœa, accompanied with vomiting. As the disease progresses, the bowel discharges suddenly become copious, consisting of a thin liquid containing a large quantity of white flocculent particles, which give to the discharges an appearance not unlike whey or rice-water; hence the name "rice-water stools" of cholera. There is entire absence of fæcal odour in the stools of cholera. The vomiting and purging now become incessant; the act of evacuation is not always accompanied with pain, but often with a sense of relief. The absence of *bile* in the vomited matter and in the evacuations, together with the marked tendency to early collapse, distinguish this disease from English or summer cholera, prevalent towards the end of the summer months and the beginning of autumn. The urine is diminished in quantity, is albuminous, or sometimes entirely suppressed, the bladder being found empty on examination after death. In some cases the first urine passed after recovery from collapse was albuminous, sp. gr. 1015–1025, and on the addition of nitric acid assumed an almost purplish black colour. Painful contractions—*cramps*—of the muscles of the soles of the feet, calves, and abdominal walls are always more or less present.

Stage of Collapse.—If the disease be not early checked, it passes into the *algide* or *cyanosed stage*, ending in fatal collapse. The temperature of the body rapidly sinks, the breathing is increased in frequency and the pulse accelerated and feeble, sometimes almost imperceptible. The skin assumes a bluish-slaty colour, and even the breath feels cold to the hand. A fall in the temperature of the body below 94·5° F. generally indicates speedy death. The face assumes a peculiar pinched expression, the eyes are sunken, the corneæ are flattened, the complexion muddy-looking, and the whole expression of the face is known as the *facies choleritica.* The secretion of milk in suckling women is not decreased, on the contrary it may be even increased. The catamenia are also not arrested. Collapse ends in death. The duration of an attack of cholera is usually from three to eighteen hours from the commencement of the symptoms. When the discharges from the bowels assume the appearance of thin gruel mixed with brick dust, a fatal termination may be prognosticated with almost perfect certainty. This condition of the stools is the result of the presence of the blood cells more or less disintegrated.

Stage of Reaction.—Should the disease, however, be arrested, the *stage of reaction*, associated with more or less febrile movement, sets in; the purging gradually diminishes, the breathing becomes slower and the pulse stronger, and the stools green and gelatinous. A rash not unlike roseola or urticaria may occur during this stage over the backs of the hands and forearms, and may even extend to the trunk. Even when this stage has most favourably set in the patient is not free from danger, as sudden collapses are by no means uncommon. During reaction Goodeve says that the urine is high coloured, acid, and possesses a strong animal odour.

Dysentery, or obstinate constipation, may follow as a sequel.

Morbid Anatomy.—There are no constant or very appreciable anatomical characteristics of cholera. The liver, brain, heart, lungs, and kidneys are generally healthy, but in some cases the organs are found gorged with venous blood. Œdema and congestion of the mucous membrane of the duodenum, jejunum, and ileum, is of common occurrence, the congestion being greatest in the mucous membrane of the ileum; not, however, extending as far as the ileo-cæcal valve by some six inches. According to Dr Church, in all the cases of cholera where death took place during, or soon after, the stage of collapse, lesions were invariably found in the alimentary canal, not unlike those of some of the irritant poisons. He would, therefore, refer the phenomena noticed in the algide stage to a shock produced by the violent effect of the poison on the intestines. Ecchymoses are invariably found on the pericardium and endocardium. The blood is usually of a "tarry" consistency and appearance, the fibrin is slightly, if at all, affected, the serum is rich in albumen, the salts are diminished, but of those which remain the potash are in excess of the soda. Due to the tremendous draining away of the watery portion of the blood, the corpuscles are apparently increased.

In most of the observed cases of cholera, there was a rapid increase in the temperature immediately preceding death, and this increase of temperature was noticeable for a short time after death. The late Dr Laycock used to recount to his class the horror he experienced one night, when, on making an incision into the abdomen of a patient dead from cholera, he found the body some hours after death quite warm, and the intestines steaming.

Pathology.—Some attempts have been made to explain the most important and characteristic symptoms of cholera. The "rice-water stools," which cannot be compared with any of the known results of inflammation, have until recently been unexplained. Thiry, by isolating a portion of the intestine of a dog, obtained a thin yellowish alkaline albuminous secretion—"*succus entericus*"—which like the pancreatic juice dissolved fibrin, but "had no action on starch" *(Foster).* Fagge states that Masloff found that the secretion so obtained "possesses the property of converting starch into sugar." Foster quoting the same authority says that "it acts feebly on starch." Be this as it may, it has been proved that when a portion of intestine has been so isolated and the nerves supplying it cut, a yellowish albuminous alkaline fluid rich in soda salts and containing mucous flocculi is obtained. Kühne has found that the rice-water liquid of cholera which so closely resembles the succus entericus possesses the property of converting starch into sugar. From the above considerations Fagge suggests that the "primary action of the cholera virus is upon the solar plexus rather than upon the bowel itself," but that the action of the intestines may be early arrested as shown in those cases of "cholera sicca," in which the intestines are found full of the fluid though none had been voided during life. The collapse of cholera may also be explained, as Cohnheim suggests, to the drain of fluid from the body and the thickened condition of the blood. It has been shown by the experiments of Sanderson and others that the fresh discharges from a cholera patient are not infective, and that they only acquire their infective properties after undergoing a process of fermentation or decomposition for two or three days, becoming again innocuous after the sixth day.

Predisposing Causes.—Of the predisposing causes of cholera little is known, the weak suffering equally with those in robust health. Starvation, improper food, excessive heat, combined with great fatigue, when long continued, may aid its development. In Paris, during an epidemic, it was noticed that the slightest operations were often followed by fatal attacks of cholera. Cholera, as before mentioned, has been known to attack the inhabitants on one side of the street, those on the other side enjoying complete immunity from the disease. This singular occurrence took place in a street in Southampton, during one of the epidemics which visited that town.

Diagnosis, etc.—This must be obtained from a study of the symptoms just given, and errors of diagnosis can only occur in sporadic cases. In India poisoning must not be mistaken for cholera, and this caution is not altogether unnecessary in England. Several fatal cases described as sporadic attacks of cholera, *cholera nostras* or *cholera Europea*, have subsequently been discovered to be due to arsenical poisoning to obtain the insurance on the lives of the persons poisoned, and which the insurance offices, on the good faith of the medical practitioner, have paid.

The **Prognosis** should be very guarded, and may be gathered from the discussion of the symptoms which presage death, and which have been before enumerated.

Treatment. — This is both prophylactic and therapeutical.

Every case of incipient diarrhœa during an epidemic of cholera should, if possible, be arrested at once, and "house-to-house visitation," conducted by properly instructed persons, should be organised. Sporadic cases of diarrhœa should be treated on the spot, and dispensaries open night and day for the free delivery of medicines; and these places might be opened and maintained along lines of traffic, railway stations, etc. Cholera hospitals should be erected, and the discharges from patients in them should in no case be allowed to enter the common sewers. All clothes should be disinfected before being washed. Carbolic acid and sulphurous acid should be used freely as disinfectants. The cisterns and drains should be examined, and disinfectants used in the latter. Quarantine has in many cases failed to prevent its introduction into a country. Popular assemblies during an epidemic should be, if possible, discontinued, and movements of troops avoided, unless absolutely necessary. The people should be instructed how to live. Ripe fruit, fresh vegetables, may be used by all, and good food supplied to the poor.

With regard to the therapeutical treatment, almost every known drug has at some time or other been named as a specific; opium, castor oil, calomel, and a host of others have all had their votaries. To arrest the primary diarrhœa there is, perhaps, no better mixture than the following:—℞ Tincturæ Opii, ʒiij; Acidi Sulphurici Aromatici, ʒiij; Aquam Camphoram, ad ℥viii. Mft. Sumat ℥i post sedes liquidas.

Dr B. W. Richardson recommends in the first stage cheerfulness on the part of the medical attendant. I agree with him that if fear does not cause cholera it predisposes to it and intensifies the symptoms. I have seen it so act. He agrees with Dr G. Johnson in the administration of a dose of castor oil, and follows its administration by a mixture, containing, creasote, ♏xii; compound tincture of camphor, ℥ss; glycerine, ℥ss; water, ℥ss. A teaspoonful in water every four hours until the vomitings and bowel discharges are relieved. In the stage of collapse, I have seen frictions to the body do good. The administration of alcohol I believe to be positively injurious. So beneficial in fever, it is here a poison, for the conditions are reversed. The fever patient has been overworked, oxidation has been excessive, the cholera patient has been underworked by prevented oxidation. (See remarks on administration of alcohol under "Typhus.") Warm, not *hot*, drinks may be given, and preferably chicken broth. Dr Stevens advocated the treatment by salines. Mixtures containing chloride of sodium, gr. 20, carbonate of soda, gr. 30, and chlorate of potash, gr. 7, were given every half-hour. Mustard plasters were used to the epigastrium, and friction to the extremities. The patient was supported by beef-tea, and the thirst relieved by soda water or pure water *ad libitum.* Opium is probably the best remedy for checking the evacuations, but must be given with great caution, if at all, on the approach of the stage of collapse. An American physician once remarked to me that, from his experience in New York, more people died from the effects of the large doses of opium, then the fashion to give, than from the cholera itself. Free diaphoresis at the beginning of an attack of cholera is often valuable. During the stage of collapse, diffusible stimulants, but not alcohol, are useful, and tepid compresses may be applied on the abdomen.

Should cholera again make its appearance in this country, a careful perusal of an article on cholera by Dr B. W. Richardson, in the *Asclepiad*, for February 1885, might lead to important results in treatment. The suggestions are well worthy of attention and practical application. The injection of saline solutions into the veins may rouse the patient sufficiently to enable him to sign a will, etc.

INFLUENZA.

Lat. *Catarrhus epidemicus.* Fr. *Grippe.* Ger. *Grippe.*

Definition.—An epidemic specific fever, marked by some catarrhal symptoms, and in some epidemics attended with considerable mortality.

In 1729 there "broke out and raged all over Europe and, perhaps, the globe, a most universal epidemic catarrh;" and "in 1732-33," says Dr Guy, quoting from Dr Short, "the most sudden and universally epidemic catarrh that has been in this age, sparing neither ranks, sexes, ages, old or young, weak or strong, and killing off many hectic and phthisical people." "In the space of twenty-five years," says Dr Guy, "four well-marked epidemics of influenza occurred, that is, in the years 1729, 1733, 1737, 1743."

This disease appears to be due to certain atmospheric changes, about which we know nothing. Several epidemics have occurred with varying mortality since the year 1510. The fatality in some epidemics has been large. Influenza of late years is seldom fatal, however, except in old people, and in those of feeble constitution. The duration of the attack is from three to six days. There are no characteristic pathological conditions found after death.

Symptoms.—The symptoms are those of specific fever of a severe type but of short duration, with severe catarrh and bronchitis. Rigors, followed by sudden and severe prostration, with depression of the spirits, are also among the principal signs of this disease.

Treatment.—In the strong and robust, the treatment consists in the free use of saline purgatives, together with diaphoretics and the careful administration of opium in the form of Dover's powder. Bleeding should never be employed, in fact no lowering measures of any kind are admissible. Complications should be carefully looked for, and treated on general principles.

DIPHTHERIA.

Lat. *Diphtheria.* Fr. *Diphthcrite.*
Ger. *Diphtherische, Entzündung der Rachenschleimhaut.*

Definition.—A specific, infectious, and contagious disease, with membranous exudation on a mucous surface (generally of the mouth, fauces, and air passages), or occasionally on a wound. To this may be added the fact that it not infrequently occurs as an epidemic. It obtains its name from *διφθέρα*, a skin.

Pathology.—The deposit of lymph, common in inflammation of serous membranes, is not usual in inflammation of mucous membranes. In diphtheria, however, it would appear that the characteristic deposit is but the local manifestation of the disease. The first local sign of the disease is a redness of the throat, but this soon passes off, and white patches are then seen here and there, which soon coalesce and cover the pharynx and posterior nares, sometimes extending even into the trachea and bronchi. This, the false membrane, as it is called, is deposited in layers, at first soft and creamy, but soon becomes firm and tough, and of a salmon colour—in severe cases changing to black. When placed under the microscope, it seems to be formed of fibres, epithelium, granules, pus, and blood globules, and sometimes the presence of fungoid growths may be detected. It is still a question whether the false membrane be an exudation, or the production of a fungus. Trousseau denied the possibility of its being due to a fungus, while Jetzerich, holding an opposite opinion, has described a fungus under the name Zygodesmus, to which he attributes the cause of the disease. Wilks, whilst admitting that some forms of throat affection attended with a white pellicle may be due to a fungoid growth, yet suggests the doubt as to these cases being true diphtheria. Some observers, Hüter and Oertel, believe that the diphtheritic contagion consists in certain forms of bacteria. The chief forms of these organisms found in diphtheria are the micrococcus and bacterium termo. These have been found not only in the false membrane, but even in the blood of those attacked by this disease. Diphtheria may be capable of being spread by inoculation, but Trousseau and others have inoculated themselves without effect. Exfoliation of the membrane is generally attended with a third or even a fourth formation of false membrane. The glands of the neck are greatly enlarged. The disease runs its course in from three to fourteen days.

Etiology.—The etiology of diphtheria is obscure. Some believe that it may owe its origin to insanitary conditions, others deny that these have any influence in its causation; but there cannot be a doubt but that it can be spread by water, and especially by milk. In the latter case the origin may be in the cow supplying the milk. A disease of the cow known as "garget" has been suggested as furnishing the poison. It is an infectious mammitis.

Symptoms.—The period of incubation varies from two to seven days. The invasion of diphtheria is not always well marked. Rigors followed by headache and some febrile movement, extreme lassitude—an important symptom—a feeling of stiffness about the neck, and difficulty and pain on attempting to swallow, are among the early symptoms. Sometimes the premonitory symptoms are so slight as scarcely to attract attention, and persons have been known to die without the presence of the disease being suspected till a *post-mortem* examination revealed the true cause of death. The tonsils then become inflamed and swollen. Sometimes deglutition is performed with little pain, even though the disease be very severe and the patient die. Pain on swallowing is therefore no criterion of the extent or severity of the disease. If the diphtheritic membrane extends into the larynx and trachea, its presence there is usually first announced by a small dry cough of a peculiar character resembling croup. The voice becomes affected, the breathing difficult, and each act of respiration produces a kind of whistling sound. The voice, as the disease progresses, may be reduced to a muffled whisper. Sometimes in the course of the disease there is remission in the symptoms, and the patient appears much better, and in a fair way to recover, when, suddenly, dangerous symptoms set in, and he dies. The knowledge that this may occur should put us on our guard about giving too favourable a prognosis.

The cause of these remissions is not very apparent, but the following have been suggested :—

1. The detachment of the membrane, and expulsion of it by coughing.
2. That the remission is a pathological phenomenon which cannot be satisfactorily explained.

The formation of the diptheritic membrane then commences as above described, and if removed grows again, but if exfoliated naturally, a fresh formation does not take place. All desire for food is entirely lost, patients often evincing the greatest repugnance to take any aliment whatever. The pulse is at times quick, at others moderate, and more or less compressible; when danger threatens, it becomes irregular. Hæmorrhage from the nostrils, often difficult to arrest, may occur. The variations in temperature are not well marked, and in many cases the temperature is scarcely above the normal. The urine is scanty, high-coloured, generally loaded with albumen during the early stage of the disease. The excretion of urea is increased. During convalescence, paralysis of the muscles which assist in the act of deglutition not infrequently occurs, the paralytic affection sometimes extending to other parts. The paralysis that frequently follows an attack of diphtheria begins in the *soft palate and pharynx*, then there is some affection of the sight, followed by paralysis of the upper and lower extremities, the muscles regaining their power in the order of their attack. Dr Fagge was of opinion that diphtheritic paralysis might be explained on "the theory of a *neuritis migrans*, a morbid process starting in the nerves of the part originally attacked, and spreading along the fibres until it reaches the centres." Death may be due to exhaustion, asphyxia, hæmorrhage, gangrene, or, as suggested by Sir W. Jenner, to the deposition of fibrin within the heart or in one of the large vessels. General dropsy and delirium are rare.

This disease is held by some to differ from inflammatory croup in four essential points—

(*a*) The local inflammation commences in the fauces, and not in the trachea, as in croup, and is liable to extend to the adjacent parts.

(*b*) By beginning as a fever, instead of a catarrh and cough.

(*c*) Croup is a sporadic affection, diphtheria often prevails as an epidemic.

(*d*) It is attended with extreme debility that rapidly comes on, and as rapidly increases, adults usually dying of asthenia, though children may be carried off by asphyxia, from the inflammation and false membrane obstructing the larynx. Whether these distinctions are more apparent than real it is difficult to say; for my own part, I am inclined to consider croup and diphtheria as *entirely distinct diseases.*

From scarlet fever diphtheria also differs in its clinical characters. The difference consists in—

(*a*) The absence of the scarlet rash, followed with efflorescence.

(*b*) Scarlet fever is rapidly developed; diphtheria often comes on insidiously.

(*c*) In diphtheria the inflammatory affection of the throat has a tendency to spread; this is seldom the case with scarlatina.

(*d*) The *sequelæ* of scarlet fever—general dropsy and albuminuria—give place to paralysis and some other peculiar nervous affections which often occur during the convalescence of diphtheria. These peculiar nervous symptoms generally begin during the second and third week after the subsidence of the throat affection. It is important to remember that *albuminuria* is a *sequela* of scarlet fever, but that in diphtheria it may be found on the second or third day of the disease. Diphtheria may also be distinguished from pharyngitis with follicular secretion, from the fact that the deposit of the latter is pultaceous not membranous, cannot be removed in stripes, and that it dips into the follicles.

The **Prognosis** should always be very guarded.

Treatment.—The primary object of our treatment consists in attempting to prevent the tendency to sudden and fatal syncope, induced by the asthenic conditions of the patient. The diet should be nutritious, consisting of milk, strong beef-tea, meat soups, and jellies. The value of alcohol in severe cases cannot be over-estimated. In some cases enemas of defibrinated blood may be used with great advantage. Ice may be given if agreeable, and it will be of great assistance in checking the constant vomiting which often occurs. The local application of the tincture of steel, or a gargle containing the chlorate of potash, will be found more valuable than the application of nitrate of silver or other caustics. Perhaps the remedies most likely to be of service are the chlorate of potash and the tincture of the perchloride of iron, both given freely, F. 25. Hot vapour inhalations, painting the throat with a solution of lactic acid or lime water, glycerine and carbonate of lime, have been suggested in the hope of dissolving the membrane. The application to the throat of a mixture of spirits of wine, carbolic acid, iodine, and water, has been strongly recommended, as have also solutions of salicylic acid, 1 to 60; chloral hydrate, 1 to 6, etc. The hydrochlorate of quinine may be used both topically and internally. The hydrochlorate is not so irritating as the acid sulphate. The benzoate of soda has been given to children of five years old to the amount of a drachm and a-half daily. Inhalations of the oil of eucalyptus have also of late been suggested. Blood-letting, local or general, together with other lowering measures are contra-indicated. As a last resort, recourse must be had to tracheotomy, and this operation should be performed before the patient gets too weak; better a little too soon than a little too late. The operator is under no necessity to suck the tube clean and endanger his own life. The performance is of very doubtful efficacy. The paralysis which is sometimes a sequel to this disease may pass off without treatment. In most cases, however, rest, tonics, change of air, are needed. Faridisation may ultimately be required, and should be used with care.

WHOOPING-COUGH.

Lat. *Pertussis.* Fr. *Coqueluche.* Ger. *Keuchhusten.*

Definition.—An epidemic specific disease, occurring most frequently among children, and characterised by a peculiar whooping noise at the end of a fit of coughing, the whoop being produced during inspiration.

Pertussis—*pertussis*, a continual cough—is a disease of early childhood, in rare cases occurring in adults. To children under five years of age it is very fatal. No anatomical appearances present themselves after death beyond those which attend ordinary bronchitis. The disease may be described as a cough, having a more or less paroxysmal tendency, often assuming a suffocative character, and attended during the inspiratory movement with the characteristic whoop. The disease is infectious, and occurs oftener as an epidemic than in isolated cases, one attack giving complete immunity from further visitations.

Symptoms.—The first stage of the disease in some cases is that of a common catarrh, accompanied with an obstinate and persistent cough; profuse expectoration of a clear viscid mucus succeeding each paroxysm. In others, the premonitory symptoms are those of bronchitis, attended with great fever, and dyspnœa. These symptoms are succeeded by the convulsive stage, with its characteristic cough. The paroxysm begins with a series of violent *expiratory* acts, which follow each other so quickly that inspiration is for a time prevented. The face becomes purple, and the eyes project and fill with tears. Just as the little patient appears on the point of suffocation, a long and difficult inspiration—the harbinger of the patient's safety—now takes place; the air as it rushes in through the half-closed glottis giving rise to the peculiar crowing sound of whooping-cough. The skin, during the fits, is sometimes bathed in a cold sweat. An attack of vomiting generally terminates the paroxysm. The child having got rid of its last meal is ready to begin another. Hæmorrhage from the nose, and an involuntary discharge of fæces, frequently take place during the paroxysm. Ulceration under the tongue may occur, and there may be temporary glycosuria, and pain in the pectoral muscles after the fits of coughing. The respiratory murmur is absent during the fits. The most frequent complications which may occur during the course of this disease, are those which relate to the respiratory organs, on the one hand—pneumonia, bronchitis, interlobular emphysema, pleurisy, capillary bronchitis, and phthisis; and on the other, the brain and nervous system—congestion of the brain, hydrocephalus, and convulsions. Though rarely fatal in itself, except in very young children, it becomes so when complicated with any of the above-mentioned affections. Whooping-cough must not be mistaken for acute bronchitis: this can only be done in the earlier stages of the disease. It must also be separated from laryngismus stridulus, and from tuberculosis of the bronchial glands at the bifurcation of the bronchi.

Treatment.—Nearly every drug in the pharmacopœia has at some time or other been declared a specific. As the curative measures appear so unproductive of good results, we may try what may be done by way of prophylaxis. Taking into consideration the epidemic character of the disease, children should if possible be removed from the infected locality, especially if they be delicate or strumous. In the early stages of the disease confinement to one room, in which a uniform temperature is maintained during the day and night, should be enjoined, and all exposure to cold avoided. A trip to the sea-side will often effect a cure when all other means have failed. If drugs be employed, belladonna, together with bromide of ammonium, or the sulphate of zinc with belladona, will most probably be found to afford the greatest relief. There is one remedy which will be found invaluable in the treatment of this disease, and that is hydrocyanic acid. I frequently combine it with the tincture of conium or belladonna and ipecacuanha, F. 29. Friction down the spine, with a mixture of the compound camphor and belladonna liniments is also useful. I speak from experience of the value of this treatment. A substitute for Roche's embrocation may be used, F. 39. During the third stage, that is, when the disease begins to decline, a meat diet, with eggs and wine, accompanied with the exhibition of ferruginous tonics, is indicated.

PAROTITIS.

Lat. *Parotides.* Fr. *Oreillon.* Ger. *Ziegenpeter.*

Definition.—An epidemic and contagious affection of the salivary glands.

Parotitis, or Mumps, is an inflammatory affection of the parotid gland, probably induced by a specific poison. Enlargement of the gland or glands, for sometimes both sides are affected, takes place, accompanied with a sensation of tenderness when touched and pain during mastication. The pain shoots up towards the ear, the lower part of which is pushed outward by the enlargement of the gland. The attack is generally accompanied with catarrhal symptoms, slight fever, and pain in the head, together with loss of appetite and general malaise. Suppuration seldom takes place, and the duration of the attack is short, with no tendency to become chronic. Towards the end of the glandular affection, the testicles in males and the ovaries in females sometimes become tender and swollen, and you may leave a patient on one day almost well, to be summoned on the next to an enlarged and painful testicle.

The disease is contagious, and occurs usually but once during life.

The **Prognosis** is favourable, though permanent deafness may result in one or both ears, probably from pressure of the swelling on the auditory nerve.

Treatment.—In most cases the administration of an active purgative in the first instance, followed by a simple saline mixture, and the application of warm anodyne fomentations to the gland, will be all that is necessary.

DYSENTERY.

Lat. *Dysenteria.* Fr. *Dysenterie.* Ger. *Ruhr.*
Syn. *Dysenterie.*

Definition.—Dysentery is inflammation and ulceration of the mucous membrane of the large intestine, most prevalent in hot climates, and often occurring as an epidemic.

Dysentery is supposed by some to be due to exposure to sudden and great variations of temperature, especially when the system is weakened by fatigue, irregular living, combined with insufficient clothing and lodging. There cannot be a doubt but that foul drinking water will give rise to dysentery. It has been the scourge of armies.

Pathology and Morbid Anatomy.—Dysentery has been divided into two forms—*catarrhal* and *diphtheritic*, but the first is merely a milder form of the latter. The inflammation of dysentery is most marked in the descending colon and rectum, but it may extend into the ileum. It is characterised by ulceration and sloughing of the mucous membrane of the bowel, varying, however, with the intensity of the inflammatory process. In the milder forms of the disease, the summits of the folds of the mucous membrane are the portions which show the morbid changes most. A greyish-white layer of fibrinous material is found covering them, which leaves, when scraped off, a slightly ulcerated surface. The solitary glands enlarge slough, and thus produce ulcers, which increase in size rapidly. In the severer forms of the disease the necrosis becomes intense, and large portions of the mucous membrane are converted into black, rotten sloughs, which in the course of time are thrown off by a sort of reactive, suppurative inflammation. Sometimes the ulcers may heal, and recovery take place. It has been suggested that the general torpidity of the lower bowel permits of the injurious delay of the poisonous material which gives rise to dysentery; or the poison may have an elective affinity for the lower bowel, as arsenic has for the stomach, and corrosive sublimate for the colon and rectum.

Symptoms.—Dysentery generally begins as a simple diarrhœa. The important symptom of this disease is *tenesmus*, or the constant desire to go to stool, accompanied with great straining and pain, but inability to evacuate the bowel, nothing being passed but mucus, blood, and shreds of fibrine, which the patient believes to be the coats of his own bowels. Small hardened lumps of fæces called *scybala* are sometimes expelled. The excessive straining at stool often gives rise to prolapsus of the rectum, and the patient may even faint, the result of the intense prostration so characteristic of the disease. The griping or colicky pains which accompany an attack of dysentery are called *torminia.* In hot climates it is a very acute disease, attended with considerable febrile movement and intense pain around the umbilicus, as well as in the course of the rectum. During the course of an attack of dysentery the patient may suffer from unhealthy-looking ulcers on the legs. They first appear as small, bluish blebs, which soon burst, and from which a sanious watery fluid pours out. They may heal to burst out somewhere else, and appear to be connected with the veins.

Dysentery has a considerable tendency to become chronic, and at the best of times it is a difficult disease to remove. The disease is not unfrequently followed by hepatic abscess.

Diagnosis.—Intersusception of the bowels, especially in male children, internal hæmorrhoids, cancer of the rectum, arsenical poisoning, etc., must not be mistaken for dysentery. Care is in all cases necessary.

The **Prognosis** in sporadic dysentery occurring in a temperate climate is favourable, but in hot countries it is a grave disease, and any opinion should be given cautiously.

Treatment.—Divers modes of treatment have been recommended for the cure of dysentery. Purgatives, calomel, castor oil, and other remedies have had their votaries. The injection of castor oil and opium has often effected a cure. The plan now generally adopted in India, and found most efficacious, consists in the exhibition of large doses of ipecacuanha.

The mode of administration is generally as follows:—Half-a-drachm of the tincture of opium may be given, followed in about an hour's time by twenty or thirty grains of powdered ipecacuanha in a little syrup. The patient is kept perfectly quiet, and he is not allowed to take any fluid, but he may suck small portions of ice. In four or five hours the dose may be repeated. Nausea and vomiting rarely occur when drink is prohibited. In chronic dysentery the extract of logwood in pill, together with the tincture of the pernitrate of iron, have been found very useful. But perhaps the most important part of the treatment consists in removing the patient to another climate and in carefully dieting him. The diet should be chiefly composed of milk and farinaceous foods.

SUB-GROUP (*b*).—*DISEASES DEPENDENT ON SOIL, CLIMATE, &c.*

INTERMITTENT FEVER, OR AGUE.

Lat. *Febris intermittens.* Fr. *Fièvre intermittente.*
Ger. *Kaltes Fieber.*

DEFINITION.—A non-contagious disease, produced by malaria and characterised by the occurrence of febrile paroxysms at stated intervals, and by the absence of fever between the paroxysms. Some enlargement of the spleen is always present.

There are three types of intermittent fever or ague, the type of the fever depending upon the length of the interval :—

Quotidian—returning daily, with twenty-four hours' interval.

Tertian—returning every other day, with forty-eight hours' interval.

Quartan—returning every third day, with an interval of seventy-two hours.

The time which elapses between the paroxysms is called the *intermission* or *apyrexia ;* the *interval* is the entire period from the beginning of one paroxysm to the beginning of the next. There are no marked anatomical lesions peculiar to this fever, if we except the spleen, which becomes enlarged from repeated attacks of the fever. The exciting causes of ague appear to arise from marsh miasma. Klebs and Tommasi-Crudeli have lately stated that from the soil and air of marshy districts they have been able to cultivate a microphyte closely resembling the bacillus anthracis, and described by them as the "bacillus malariæ." They assert that by inoculation with these they have produced ague in rabbits. It is also stated that the bacillus may be found in the spleen of men dying during an attack of ague. Dr Salisbury of

Cleveland, U. S., holds that he has discovered the cause in the sporules of a species of palmellæ. Be these conjectures correct or not, it is well known that the effluvia from decomposing vegetable matter is most active, especially when the land is drying after being soaked with moisture, as in the spring and after the early autumn rains. The poison is more virulent at night, and generally spreads in the line of prevailing winds, hugging, as it were, the ground, seldom rising to any considerable elevation. Hence rooms in the upper storeys of houses in malarious districts should be used as bedrooms. Groves of trees, or running water, appear to intercept its progress. A temperature of at least 60° F., it would seem, is required for the generation of *malaria*, its evolution being checked when the thermometer stands at 20° F. Malaria may be transported to considerable distances by atmospheric currents. A certain state of acclimatisation seems possible, and negroes suffer less from the poison than Europeans. One attack of ague rather increases than lessens the susceptibility of the poison. The period of incubation has not yet been determined.

Symptoms.—In the majority of cases an attack of ague comes on suddenly; premonitory symptoms when present are not very distinctive of this disease.

The paroxysm consists of three stages—the cold, the hot, and the sweating.

In the *cold stage*, there is chilliness, shivering, chattering of the teeth, pallor, and contraction of the features. The skin presents the appearance known as "goose skin." The patient complains of aching pain in the back and limbs, accompanied with a feeling of oppression at the chest, causing sighing and yawning. The pulse is frequent, but small and feeble, the tongue white, sometimes furred. The urine is pale and abundant, and there is frequent desire to pass water. The commencement of a paroxysm, notwithstanding the sensation of coldness experienced by the patient, is most frequently indicated by a sudden, decided, and rapid rise of temperature, the high temperature being maintained during this stage 103° to 106·3° F. In the cold stage the blood leaves the surface and accumulates about the liver, spleen, and bases of the lungs, and the right side of the heart. The duration of this stage varies from four or five minutes to as many hours.

In the *hot stage*, generally lasting from two to eight or ten hours, the skin becomes hot and the face flushed; the patient complains of great thirst and throbbing headache. The tongue becomes dry, the pulse quick, full or bounding ; the urine is still abundant and loaded with urates, and of a high colour. The thermometer placed in the axilla indicates a temperature usually from 105° to 106° F.

In the *sweating stage*, perspiration breaks out upon the forehead and face, and then on the body and extremities. The heat of the surface is diminished, the thirst and restlessness cease, and the patient feels relieved. The thermometer marks a rapid defervescence and return to the normal standard. The pulse subsides, the urine is scanty in quantity, and deposits urates of soda and ammonia.

In the hot stage the heart reacts and throws out the blood again to the surface, but with too great violence, and the secretions are stopped. In the sweating stage, however, the secretions are restored and the balance of the circulation reproduced.

The febrile paroxysms vary considerably, as before shown is the case in the different stages. The whole paroxysm may be over in one or two hours, or may last for eight or ten hours. The paroxysm of the *quotidian* occurs early in the morning, that of the *tertian* about noon, and that of the *quartan* in the afternoon between three and five o'clock. Of the three primary types, the quotidian and tertian are by far the most prevalent in Europe; the quartan variety is comparatively rare.

The quartan is said to have the longest cold stage, the tertian the longest hot stage. A rare variety called the *double tertian* sometime occurs, in which there is a fit daily, but only those on alternate days resemble each other and take place at the same hour.

Owing to the repeated internal congestion during the cold stage, the liver and spleen become more or less affected; the latter organ especially greatly increases in size, and is then commonly known as "ague cake" in malarial districts.

The belly is often swollen and tumid, and the countenance assumes a yellowish or sallow tint, with more or less œdema, giving rise to what is called the *malarious cachexia.*

Persons suffering from malarious symptoms are very liable to contract pneumonia, often of a very severe character. It may here be noticed that some diseases may assume an intermittent character; thus, hectic fever, symptomatic of phthisis or abscess in the liver, or from any other cause, may assume an intermittent type. Hectic fever, however, more closely resembles remittent fever than ague. Women soon after delivery may be attacked with an intermittent form of fever, generally yielding to valerian and other spasmodics. It is well to remember these facts, or a case of incipient phthisis or abscess of the liver may be treated as a case of ague. Certain forms of fever, accompanying disease of the brain and urinary system, often assume an intermittent type.

Ague is seldom fatal when uncomplicated.

Treatment.—The treatment of intermittent fever is divided into two parts, palliative during the paroxysms, and curative during the intermissions.

During the cold stage the patient should be put to bed, and every attempt made to restore warmth to the body; hot tea or coffee may be given, and friction applied to the extremities. Relief during the hot stage is gained by sponging the body with cold or tepid water.

Ice or iced drinks are indicated to relieve the thirst. Careful protection from cold draughts, and wiping the perspiration from the body with warm flannels, will promote the comfort of the patient during the sweating stage. The curative measures consist in the administration of a brisk cathartic, followed with the exhibition of large doses of quinine. The most successful mode of procedure appears to be as follows:—

Ten or twenty grains of the sulphate of quina should be given in solution during the sweating stage, followed by grain or two-grain doses three times during the day. Some recommend the administration of quina in five-grain doses till cinchonism is produced, and continued in smaller doses for some weeks. Roberts advises the administration of gr. iii.–iv. every four or six hours during the intermission. It has been noticed by Graves and others, that the administration of quina will often change the type of the fever, and that when this is the case, the liquor arsenicalis will, in many cases, cure the disease. Arsenic may be given at any stage of the fever. Fowler's Solution is the favourite "ague drop" of the Fen districts. For the treatment of the congestion of the liver and spleen, so generally present in this fever, leeches may be applied to the anus, imitating Nature's method of relieving intestinal congestion by bleeding piles. As long as there is tenderness over the liver, Graves recommends the use of mercury to keep the bowels open, and then iodine, alternately, with vegetable and mineral tonics. The enlargement of the spleen may be treated by the external application of the binoidide of mercury ointment. The anæmia which is often a sequel to ague is best treated with chalybeates. Salicin, arsenic, the hydro-chlorate of ammonia, and a host of other drugs, have been lauded as cures for ague.

Of course no treatment can be expected to give permanent relief so long as the sufferer remains in a malarious district.

REMITTENT FEVER.

Lat. *Febris remittens.* Fr. *Fièvre remittente.*
Ger. *Bösartiges indemisches Fieber.*

Definition.—A malarious fever, characterised by irregular repeated exacerbations, the remissions being less distinct in proportion to the intensity of the fever. It is accompanied with functional disturbance of the liver, and frequently by yellowness of the skin.

Paludal, Marsh Fever, Bilious Remittent Fever or Remittent Fever.

A malarious fever characterised by the occurrence of remissions instead of intermissions, as is the case with the fever just described. The remissions are less distinct in proportion to the intensity of the fever. Remittent fever is most common in hot climates, but especially so on the coasts of Africa, about the mouths of large rivers, where the marsh miasm is very virulent.

The anatomical changes characteristic of this fever are found in the spleen and liver. When death occurs, the liver on examination shows signs of softening, and presents a slate, chocolate, or bronze colour. The colour is due, it would appear, to a deposit of pigment in the hepatic cells, or in the capillary network of the portal and hepatic veins. The spleen is more or less enlarged, softened, and contains a like deposit of pigment.

Symptoms.—The premonitory symptoms are those common to most fevers. The febrile paroxysm may last from six to forty-eight hours, and then subside, but does not disappear—there is a *remission*, not an *intermission.* During the fever, vomiting of a greenish or yellowish fluid may occur. Typhoid and Remittent Fever may be present simultaneously in the same patient; in that case the symptoms of each are mingled together. The duration of the disease is generally a fortnight.

Treatment.—The first thing to be done by way of treatment is to remove the patient from the malarious locality. The same medicines, with their mode of administration, as recommended in the case of intermittent fever, are applicable in this disease. Warburg's tincture has much reputation in this affection.

SUB-GROUP (*c*).—*SEPTIC DISEASES.*

ERYSIPELAS.

Fr. *Erisipèle.* Ger. *Erysipelas* or *Pothlauf.*

DEFINITION.—Inflammation of the integument, tending to spread indefinitely. May also spread into the subcutaneous cellular tissue. Varieties—(*a*) Simple or cutaneous. (*b*) Phlegmonous or cellulo-cutaneous. (*c*) Diffuse inflammation of cellular tissue.

Erysipelas (from Ερύω, to draw, and πελας, near; or from ερυθρὸς, red, and πελλος, livid) is a disease characterised by fever and the appearance of a peculiar eruption on the skin. The inflammation which also attends the eruption has a tendency to spread and to invade the areolar tissue. In Scotland it is known as the *Rose*, and in England it is sometimes called *St Anthony's Fire.*

By some erysipelas is classed among the eruptive fevers; by others it is held to belong to that class of diseases the result of a morbid condition of the blood, due to the inception of a specific poison, of which pyæmia is the type; and yet a third party claim that it is simply a local disease. The last of these three views appears to be founded on the fact, frequently noticed by surgeons, that when the disease becomes as it were epidemic in a ward, those patients are most frequently attacked who have an open wound, and that it is about the wound that the first appearance of the disease is noticed.

Now this, though true as a rule, is not invariably the case, and it has been observed that parts of the body have been attacked at some distance from a wound, the parts around which have entirely escaped. Moreover, an attack of scarlet fever or measles sometimes follows a surgical operation, and when such is the case, the disease almost invariably commences at the seat of the wound, and is no proof of its local origin or local nature. The first and second views demand more serious attention.

In the first place, erysipelas has many points in common with the eruptive fevers; it has a period of incubation, varying from three days to a week; the eruption on the skin is almost always preceded by marked general constitutional disturbance, and is accompanied with fever; the disease runs a tolerably regular and definite course, passing away after a limited duration; it is infectious; one attack does not, however, act as a preventive to subsequent ones.

In this last particular it differs materially from the eruptive fevers, one attack of which, except in rare instances, granting future immunity from the disease. With pyæmia it has many features in common, the most important of which is the fact that in fatal cases the blood is found greatly disorganised, the internal organs congested, and the presence of pus always detectable in the smaller pulmonary vessels.

Dr Russell Reynolds says:—"It is well known that in many cases of fatal erysipelas, evidences of disease may be found in the spleen, liver, kidneys, lungs, bronchi, larynx, trachea, and fauces; but there is nothing specific in the character of the changes discovered in these organs—nothing, that is, which is peculiar to the disease called erysipelas; nothing, indeed, which depends on erysipelas *per se;* but all that may be found is only the sign of such general blood-change as may be associated not only with the disease now under consideration, but also with that large group of maladies which stand in close relation with pyæmia."—*System of Medicine,* vol. I., p. 687.

Symptoms.—The patient about to suffer from an attack of erysipelas generally complains, for some days before the disease makes its appearance, of feeling unwell and "out of sorts." There is nothing, however, very pronounced to make him suspect the nature of the approaching attack, till perhaps he may be suddenly seized with a shivering fit, rigor, and vomiting, with a burning, smarting pain about the part where the eruption first makes its appearance. Sore throat is an early and invariable accompaniment. The constitutional derangement now becomes intensified; the pulse is quickened, the feverish symptoms increase, and the temperature rises rapidly, and may reach in a few hours 104° F. or more. As long as the inflammation is spreading the temperature continues high, even as much as 106° F. The defervescence is sudden, but is liable to sudden and abrupt rises and falls before convalescence is well pronounced. The parts attacked by erysipelas become red, hot, swollen, and painful, with a peculiar indurated feel when pressed by the finger; in some cases large bullæ are formed, which may ultimately burst and dry up, or from which a constant oozing may take place for some time before desiccation sets in, followed by the peeling-off of all the skin of the part affected. The face and those parts of the body which are most exposed are usually the seat of the disease. When the face and head are affected, the inflammation may commence by a small patch on the side of the nose, but it soon spreads and the features become distorted, the eyelids swollen and closed; the ears often lose their shape and have the appearance of fleshy appendages, with here and there a small blister, or, in some cases, one large one, from which a watery discharge continually oozes. In fatal cases the result may be due to exhaustion, to an extension of the inflammation to the membranes of the brain, or to the glottis; death in the second case being preceded by coma as a result of effusion; in the third it may be due to suffocation. When the body is attacked I have seen an extension of the disease to the lungs prove fatal, the *post-mortem* examination revealing an extensive pyæmic condition of the entire lung substance. During the course of the disease the patient is not unfrequently delirious; the delirium, becoming worse towards night, may be low and muttering or noisy and violent, and is probably not due to inflammation of the brain, but to an altered condition of the blood and of the nervous system.

Erysipelas may be either *idiopathic*, when it arises from internal causes, or *traumatic*, when it follows wounds.

The disease has been divided into three varieties—

1. Simple.
2. Phlegmonous; the inflammation extending into the subcutaneous tissues.
3. Diffuse; in which the inflammation has a tendency to spread indefinitely over the surface of the body.

Erysipelas may terminate in three ways; by resolution, by vesication, or by gangrene. During the course of the disease the urine may become albuminous, and there is always a diminution in the quantity of the chlorides.

The **Prognosis** should always be guarded, especially in the very young and the aged. The return to health is marked by a gradual fading away of the redness, and by a like disappearance of the swelling and constitutional disturbance.

Causes, etc.—Of the specific nature of the poison of erysipelas nothing is known; but the predisposing causes would appear to be a full habit of body, constitutional peculiarity, previous attacks, the period of early infancy or of middle age (from 20 to 40), the female sex especially during menstruation, extreme debility, intemperance, and bad hygienic conditions as to overcrowding, ventilation, and improper food. Sometimes the attack appears to be the result of sudden exposure to cold. It spreads chiefly by contact, may be inoculated, and is conveyed by fomites. It often prevails as an epidemic, and when introduced into a ward of an hospital, patient after patient may be attacked, necessitating the closure of the ward for a time. For this reason the old "Dreadnought" hospital ship had to be broken up, and an hospital on shore substituted.

Treatment.—The treatment consists in the adoption of measures to reduce the inflammatory action, to support the system when necessary, and to protect the inflamed parts from contact with the air. The first indication is carried out by the administration of a brisk saline purgative, and this is particularly necessary in cases of erysipelas occurring in strong and plethoric individuals. With the first few doses of the purgative, small doses of the vinum antim: tart: may be advantageously combined. And if the patient be strong and lusty, I continue this treatment for two or three days. Having emptied the bowels, I then give the tincture of the perchloride of iron in ten or fifteen-drop doses every four hours, and order the part affected to be dusted thickly over with flour from a dredger. The diet during the first few days should be light and unstimulating, consisting chiefly of milk, beef-tea, and sago or tapioca puddings. Barley water, acidulated with lemon juice, forms a very pleasant drink. But in the treatment of this disease, as of every other, no hard and fast lines can be drawn. The constitution of the patient must be considered, and must in all cases regulate our treatment. If there be a suspicion of gout, colchicum may be advantageously combined with the other remedies, and in those cases where the tincture of iron is not well borne by the stomach, quinine, or the tincture of bark and ammonia, may be substituted. A combination of the tincture of iron and the liquor ammoniæ acetatis, will often be taken when the pure tincture is rejected. Wine may be given from the commencement of the attack, if the patient be weak and debilitated: a line of treatment advocated by the late Dr Robt. Williams, irrespective of the symptoms and the part affected. The sleeplessness too often present in these cases is best relieved by the administration of the hydrate of chloral, or the extract or tincture of hyoscyamus. Pencilling round the inflamed parts with a stick of nitrate of silver, the application of collodion, or of the tincture of iodine, has been recommended by some; a lotion composed of the sulphate of soda in water is advocated by others. Hot poppy fomentations may be tried, but I have invariably found the application of flour most agreeable to the patient.

In the phlegmonous form of the disease, deep incisions must be made in the part, and a large warm linseed-meal poultice applied.

Great care must be taken by doctors and nurses to avoid carrying the disease to others.

SEPTICÆMIA AND PYÆMIA.

SEPTICÆMIA.

Lat. *Septicœmia.* Fr. *Septicémie.* Gr. *Septicaemie.*

DEFINITION.—Blood poisoning, by the admission into or generation within the body of a poison, the exact nature of which is unknown, accompanied with severe febrile symptoms and, as a rule, terminating in pyæmia and death. Leaving, when unaccompanied with pyæmia, no characteristic *post-mortem* signs.

PYÆMIA.

Lat. *Pyœmia.* Fr. *Pyohémie.* Ger. *Pyœmie.*

DEFINITION.—A severe and dangerous febrile affection, due to the entrance of a peculiar poison into the body or formed within it, and resulting in the formation of metastatic abscesses in the viscera and other parts.

Pyæmia and Septicæmia appear to result from the absorption of some poisonous matter or other into the blood. Septicæmia may be taken as the general term for blood poisoning or putrid intoxication; pyæmia where, together with the general symptoms of blood poisoning, we have outward and inward manifestations of the action of the morbific material. In describing the condition of the system known as pyæmia we also describe septicæmia, plus the above-mentioned manifestations of the poison. The exact nature of the poison is unknown, but it would appear to be more or less connected with inflammatory products which have undergone putrid decomposition. Some have tried to connect the poisonous infecting matter of pyæmia with the presence of *bacteria*, and of these Dr Burdon Sanderson has described two forms, one rod-shaped, endowed with the power of spontaneous movement *(bacterium vibrio)*, and the other dumb-bell shaped, and incapable of movement *(bacterium varicosum)*. Mr Savory has shown that by the introduction of animal matter undergoing active putrefactive change, the decomposition of animal matter thus impregnated is hastened. As a rule, pyæmia occurs in cases where there is an open wound, but it may also be present in those where no such external wound is present. Thus, it may result from necrosis of bone without external wound. In these cases, as in the others, the poisonous matter is most probably absorbed by the veins and lymphatics, and by them carried to the various parts of the body in which we find abscesses formed. These abscesses are usually of *embolic* origin, a good instance of which may be found in abscesses of the liver secondary to an attack of dysentery.

Morbid Anatomy.—In septicæmia there are no well marked *post-mortem* signs, but there is a tendency to rapid putrefaction. In pyæmia we have the presence of abscess more or less circumscribed in different parts of the body.

Symptoms.—From eight to ten or fifteen days after the infliction of a wound, a sudden rigor or shivering fit takes place, followed by cold sweats. The colour of the skin becomes muddy and sallow, and the patient drowsy or delirious—the delirium being at times noisy, at others low and muttering. The respiration is laboured, and the countenance anxious, the tongue dry, the abdomen swollen, and there may be constipation or diarrhœa. The pulse is small and wiry. The joints and tendons become painful, and metastatic abscesses make their appearance in various parts of the body, most frequently in the liver, where they may be found after death, just under the peritoneal covering. These abscesses may also be found in the lungs, kidneys, and spleen. During these changes the wound becomes pale, and unhealthy granulations are formed; the pus is sanious and watery, and sometimes an erysipelatous blush forms round the wound.

Treatment.—Isolation from other patients in the ward, if the case occurs in hospital. Let the diet be nourishing, and give tonics—quinine, iron, and iodide of potassium. Good ventilation and cleanliness are absolutely necessary. Treat secondary abscess with poultices, and free opening as soon as pus is fully present.

PUERPERAL FEVER.

Lat. *Febtis puerperarum.* Fr. *Fêbre puerperale.*
Ger. *Puerperalfieber ; Kindbettfieber.*

Definition.—A continued fever, communicated by contagion, occurring in connection with child-birth, and often associated with extensive local lesions, especially of the uterine system.

Opinions vary as to the pathology of this disease, and nothing very definite has been as yet decided with regard to its cause and nature. Some have attempted to point out its close relationship to surgical fever, others have endeavoured to show its connection with pyæmia, scarlet fever, erysipelas, and typhus fever. Certain it is, that puerperal fever will frequently make its appearance, with all its terrible fatality, in connection with the infection from any one of the above diseases, but especially from erysipelas, carried by the medical attendant. Puerperal fever was known and described by Hippocrates and Avicenna, who considered it to be due to inflammation of the uterus. The character of the disease differs with each epidemic, and it has been found also necessary to vary the methods of treatment. Several forms of this disease have been described; thus we have the inflammatory, the gastro-bilious, the epidemic or contagious typhoid, the adynamic, the ataxic or nervous, and so on. Some consider the disease to be first a local affection—peritonitis—with symptomatic fever; others that it is a specific fever. Perhaps the best and most correct course to adopt is to consider the disease *as a form of septicæmia manifesting itself in puerperal women.* We would thus drop the doubtful term *puerperal fever*, and substitute for it that of *puerperal septicæmia* as a general term for the disease, naming its local forms as puerperal peritonitis, etc.

Morbid Anatomy.—The signs of pyæmia are most frequently present. The uterus may be soft, with a most offensive discharge, and abscesses may be found in various parts of the body, lungs, etc.

Symptoms.—The attack generally begins four or five days after delivery. The symptoms will vary with the part locally affected; thus, if peritonitis be present, all the symptoms characteristic of that affection will be more or less present. First in order, perhaps, is an attack of rigors, with suppression of the lochia, then pain in the region of the womb, hot dry skin, great thirst, and a quick irregular pulse of 100 to 120, small and compressible. The heat of the skin soon subsides, and during the course of the disease the thermometer may mark no great divergence from the normal standard. Nausea and vomiting are frequently present, with considerable headache. The abdomen is swollen and distended with wind. Respiration is quick and difficult. A clammy offensive sweat breaks out over the patient, who gradually becomes weaker and weaker, and ultimately dies of exhaustion, some slight low muttering delirium being present before death. Should she recover, however, her convalescence is prolonged from the intensity of the previous exhaustion, and she may remain feeble for many months.

Treatment.—Place the patient in a well-ventilated airy room, and apply hot fomentations and poultices to the abdomen. The bowels to be gently relieved by castor-oil. Opium, to relieve pain, may sometimes be combined with small doses of mercury. The uterus may, in the first stages, be syringed out with a weak tepid solution of Condy's fluid, or a lotion of carbolic acid, one part of the acid to sixty of water. Stimulants when necessary, bark and quinine, and a nourishing diet, will form the best aids to recovery. Rest in bed and perfect quiet must be enjoined.

SUB-GROUP (*d*).—*VENEREAL DISEASES.*

SYPHILIS.

Lat. *Syphilis.* Fr. *Syphilis.* Ger. *Syphilis.*

The origin of syphilis is held by some to date from the return of Columbus from the West Indies, where his sailors are supposed to have contracted the disease. "Remembering also," says Draper, "the special circumstances under which, in this disease, that contagion is imparted, the rapidity of its spread all over Europe is a significant illustration of the fearful immorality of the times. If contemporary authors are to be trusted, there was not a class, married or unmarried, clergy or laity, from the holy father, Leo X., to the beggar by the wayside, free from it."

Pathology, etc.—Syphilis is the type of a truly contagious constitutional disease, *direct contact* with the virus being necessary for its origin and spread. It is propagated by direct contact with a primary sore, by kissing, by nursing, as in the case of wet nurses, and it has even been maintained that the semen of an infected man injected into the vagina of a healthy woman, irrespective of pregnancy, may propagate the disease. Not only is syphilis propagated by direct contact, but it may also be inherited. It is considered by some to be a specific fever accompanied with local eruptions of varying character, and that the sore is "not a necessary antecedent to infection." One thing is certain, that unlike other animal poisons which run definite courses and then exhaust themselves, syphilis may, after an apparent absence from the infected individual, again make its appearance in one or other of the forms of disease to which a syphilitic origin is attributed. There are several stages of this disease, known under the terms primary, secondary, and tertiary; the *first* when the disease is limited to the part inoculated and the lymphatic glands connected with it; *secondary*, when it affects parts not directly inoculated; and, *tertiary*, when after an interval of apparent health the disease returns with one or more of its recognised manifestations. Constitutional syphilis of the child, derived during fœtal life from one of the parents, is known as hereditary syphilis. If we compare syphilis with the exanthemata—small-pox or measles—we may consider that the secondary stage of syphilis corresponds to the stage of general blood-poisoning in those diseases, while the tertiary stage is the analogue of the period of sequalæ.

Symptoms.—After an uncertain period of incubation from the last unclean coitus, or otherwise inception of the poison, the patient complains of a slight hard and indurated sore, generally under the foreskin, but sometimes on the body of the penis. After a few days the lymphatic glands of the groin become enlarged, accompanied with more or less disturbance of the general health. The enlarged glands are not painful and seldom suppurate. The secondary symptoms may not appear for several weeks or months after the healing of the primary sore, and are then ushered in by well marked febrile disturbance, and often with neuralgic pains in the head—"bi-temporal neuralgia"—and in other parts of the body. A papular eruption, unaccompanied with itching, may now appear on the body. The throat then becomes sore, and ulcers form on the soft palate and tonsils. Unless checked by treatment, the following affections may now make their appearance:—warts, ulcers, and eruptions having a specific character, may appear on the skin; the hair may fall off and alopecia result. The bones may also become affected, and the most painful of these affections are often to all appearance merely slight periosteal swellings. The pain is most severe at night, and according to Bäumler, is due to the determination of blood to the affected parts. This determination of blood to the local periostitis accompanies the febrile rise in temperature, which frequently takes place at night. Gliding almost imperceptibly from the second stage, and carrying with it many of the affections of that stage, the tertiary commences. The general health becomes more and more deteriorated, and severe complications now arise, there being a marked tendency to the formation of tumours in the several organs of the body. The tumours on account of their gummy, semi-fluid contents are known as "*gummy tumours*," or, shortly, "*gummata*." The internal organs most frequently attacked by them are the liver, testicles, and brain. They are not of the nature of an exudation, but appear to be composed essentially of "that cytogenous or adenoid tissue which is often formed in chronic inflammation," the starting point of the new growth being the connective tissue or vessels, "never glandular or specialised elements."

The symptoms to which these tumours give rise will depend on the organ in which they are located.

The eruptions on the skin accompanying constitutional syphilis present certain peculiarities. They have a tendency to become circular or semicircular, seldom angular. Those belonging to the *secondary stage* have a *bilateral tendency*, are superficial, and do not, as a rule, leave a scar; they are often polymorphous —that is, we may have pustules, papules, and vesicles side by side, and these may assume a coppery hue. In most cases the eruptions do not itch. The eruptions of the *tertiary stage* are non-symmetrical, attack the deeper structures, which they destroy, and leave permanent scars. I have seen on the penis an induration like cartilage in the place of a former syphilitic sore. It had made its appearance at varying intervals, and had spontaneously disappeared, but owing to its longer duration I was consulted. The cartilaginous nodule entirely disappeared under a mild mercurial course of hydrargyrum cum cretâ. This affection was first described by Mr. J. Hutchinson.

Prognosis.—Although syphilis is not directly fatal in itself it may cause death by disease of the liver, the patient dying dropsical; by phthisis; by disease of the arteries ending in apoplexy; by disease occurring in any part of the nervous system giving rise to fatal nervous diseases, or by a general break up of the constitution.

Treatment.—The primary sore should be at once destroyed with strong nitric acid, and the black or yellow wash used freely to the part. Mercury by one of the four following methods should now be administered: inunction, fumigation, internal administration, or by hypodermic injection. The treatment by inunction is by no means a cleanly process, and in some persons with very delicate skins the inunction of mercury may produce troublesome and extensive patches of eczema. In cases where mercury is badly borne by the stomach, inunction is of much service. From fifteen to thirty grains of the blue ointment may be rubbed in daily, a different part of the body being selected for each day's inunction, care being taken to avoid the hairy parts of the body from the tendency of these parts to become affected by eczema. The treatment by fumigation consists in subliming eight to fifteen grains of calomel in a properly constructed apparatus, so that the patient's body is exposed to the mercurial fumes. The fumigation should be made at night, just before the patient retires to rest, and should be limited to fifteen or twenty minutes. Messrs Allen & Son, of London, manufacture a most convenient and very moderately priced apparatus for this purpose, and as it combines a Turkish bath as well, it is useful for other purposes than mercurial fumigations. The fumigations may be continued for two or three months, a slight affection of the gums being deemed sufficient evidence of the required mercurial action. For internal administration the preparations of metallic mercury, such as the blue pill or mercury and chalk, are the best. The preparation I prefer is the hydrargyrum cum cretâ, three to five grains, given night and morning, with a quarter of a grain of powdered opium. In hereditary syphilis, small doses of mercury and chalk are most serviceable. The corrosive sublimate may also be used with advantage in many cases, but should be given after meals, to prevent its disagreeing with the stomach. A solution of corrosive sublimate may be used hypodermically, but it is not unfrequently followed by abscesses in the part of the body punctured. During all the mercurial courses mentioned, strict hygienic rules must be adopted, and patients prevented from taking cold. Sometimes it is well to recommend a change of residence, and to discontinue the medicine for a time. Syphilitic anæmia is often benefited by mercury. The iodide of potassium is most useful in syphilitic affections of the bones, and in rheumatic attacks which not unfrequently occur during the course of syphilis, and in hereditary syphilis. It may also be given with marked advantage in tertiary syphilis. The iodide may be advantageously combined with the carbonate of ammonia or the aromatic spirit of ammonia. Grey powder is of great use in infantile syphilis. In warty growths round the anus the yellow wash is invaluable, and for these it is far more effectual than the black wash.

SUB-GROUP (*e*).

FEBRILE DISEASES COMMUNICABLE FROM ANIMALS TO MAN.

RABIES.—HYDROPHOBIA.

Lat. *Hydrophobia.* Fr. *Hydrophobie.* Ger. *Hundswuth.*

DEFINITION.—A disease produced in the human subject from the bite of a rabid animal, accompanied with spasm of the muscles, delirium, and an intense dread of water.

Rabies is a disease common to many animals, especially those of the family *Canedæ*, of which the wolf and domestic dog are types. It probably never rises spontaneously among these animals, but is conveyed by inoculation through the bite of an animal affected with the disease. Lawrence states that Dupuytren injected the blood of a rabid dog into a healthy one, but without producing the disease. The saliva, therefore, appears to alone contain the poison capable of reproducing the disease in man or in the lower animals. It has been suggested that heat favours the production of the disease, but this suggestion does not appear to be well-founded, and it is remarkable that rabies is rare in the West Indies. The French also observed during their residence in Egypt that the complaint was not known there, either in the canine or in the human species. The weight of evidence, therefore, is against its spontaneous origin either in man or other animals, and in favour of its communication by the saliva of a rabid animal.

Lawrence mentions the following experiment performed by Magendie and Buschet:—"They took the saliva of a patient labouring under hydrophobia in the Hôtel Dieu, and applied it to the recent wounds of two dogs: they did this on the 19th of June. On the 26th of July, one of the dogs thus inoculated went mad, and that dog bit two others, one of which died rabid on the 26th of August; so that if this experiment be correct—and the high reputation of those who are said to have made it lead us to place credit on the statement—we must, I suppose, admit that the saliva of the human subject has the power of communicating the disease." The period of incubation varies from thirty days to two years or more. It more closely resembles syphilis than any other disease in the irregularity of its appearance after the period of inoculation. Recovery from hydrophobia seldom, if ever, occurs. The word hydrophobia has been used when the disease attacks the human species. The term is a bad one, when applied to animals, for the word hydrophobia (from υδωρ, water, and φοβος, dread) merely marks a symptom of the disease when it occurs in man, and is by no means characteristic of rabies in any of the lower animals.

Symptoms.—In the Dog.—There is no aversion to water or fluids, for the dog will drink water; in this particular, rabies in the dog differs from the disease when inoculated in man. Symptoms of rage or fury are also absent. The dog is sullen and peevish, taking little notice of his surroundings. He will leave his home, and run wildly about, but will not go out of his way to attack other animals or man, biting only at those that may cross him. As he becomes worse he will pick up and eat bits of straw or dirt, and if tied up will grow angry and gnaw at the chain or any object near him. From his jaws a continuous stream of saliva flows.

In Man.—In some cases, within four to six weeks from the bite of the rabid animal, the patient complains of being unwell, and of feeling a pricking, tingling sensation in the region of the wound, if it is still open. Sometimes, when the wound has not healed, it may become angry-looking, swollen, and inflamed, but in the majority of cases, the wound has healed, and the patient has forgotten all about its occurrence. Vesicles, it is said, are also in some cases found under the tongue. In most cases, however, the peculiar symptom of hydrophobia—dread of water—soon shows itself. When the disease has become pronounced, it may be divided into three periods:—(*a*) The Melancholic. (*b*) The Hydrophobic, or Stage of Excitement. (*c*) The Stage of Paralysis, or of Asphyxia.

(*a*) *The Melancholic.*—The patient complains of coldness and shivering; his countenance is pale, anxious, almost terror-stricken, and he is troubled with terrible nightmares: his respiration is jerky and irregular, and he complains of a sense of restriction in the region of the heart. He speaks disjointedly and in a rambling, incoherent manner. The appetite is lost, and vomiting is not unfrequently present. The duration of this period is from two to three days.

(*b*) *Stage of Excitement.*—The disease now becomes pronounced. The sufferings become intense, the horror of liquids and the raging thirst increase as the disease progresses, and he is continually engaged in spitting the saliva which collects in his mouth. To assuage his thirst he attempts to drink, but in spite of every apparent effort of the will to overcome his dread of fluids, the moment he tries to drink he becomes convulsed or delirious. Convulsive attacks now make their appearance, and, if a male, satyriasis, if a female, nymphomania, may be present. Every attempt to drink only increases the severity and frequency of the convulsions. There is constant desire to pass water; the bowels are confined. The pulse is quick, but the temperature of the body is not materially increased. At times there is delirium, wild and furious; at others, low and gentle. The duration of this stage is from one to two days.

(*c*) *Stage of Paralysis and Asphyxia.*—The patient becomes weaker and weaker, cold clammy sweats break out over his body, the saliva becomes more viscid and tenacious, the eyes are suffused and dejected, the convulsions increase in frequency, but decrease in strength; he becomes paralysed, and either dies asphyxiated or comatose. Hydrophobia, as a rule, is fatal in from thirty-six hours to four or five days.

Pathology.—Once supposed to be a form of tetanus. Mouth, tongue, and pharynx inflamed. Internal organs congested, blood fluid, and of a dark colour. Brain and spinal column congested. The glands at the back of the tongue enlarged and covered with secretion. A late discussion on hydrophobia at the Royal Medical and Chirurgical Society has thrown but little, if any, light on the nature of this disease. There was a learned talk on leucocytes, blood vessels, and nervous system, but little else. Appearance of the face, that of death from strangulation.

Treatment.—*Local*—Immediately after the infliction of the wound from a bite of a mad dog, cut the part freely out, suck or well wash it, and apply nitric acid or other caustic. The simple application of nitrate of silver to a bite from a dog may amuse the patient, but it will in no way protect him from an attack of hydrophobia.

General—Hot-air baths, bromide of potassium, chloral, chloroform inhalation, subdermic injection of morphia, Calabar bean, etc. Of late years M. Pasteur has claimed that he can prevent an attack of hydrophobia by inoculation of the attenuated virus from a rabid animal. But when we consider the uncertain duration of the stage of incubation I think we must wait a more lengthened trial of his treatment before a positive opinion can be given of its efficiency.

THE FOLLOWING TABLE IS TAKEN FROM HOLMES' "SYSTEM OF SURGERY."

	HYDROPHOBIA.	TETANUS.
General Character.	Spasms of muscles of brief duration; if not voluntary, at least temporary, and ceasing to exist during intervals of rest and quietude, the jaw being relaxed, and opening and shutting regularly. The spasms are clonic.	Spasms of muscles more continued, less remitting, and more intermitting; constant rigidity of the muscles of the jaw, becoming gradually fixed and closed; tonic spasm. The cause is exposure to cold, or a wound; it rarely arises from the bite of an animal, and generally occurs soon after the injury. The bite of a tetanic animal does not produce tetanus.
	Vomiting and gastric pains general; mind subject to rabid influences and numberless deviations, passing to delirium; intolerant sensibility of surface and organs of sense.	Vomiting and gastric pains rare; mind generally clear to the last.
Countenance.	There is an expression of excitement, fearful distress, and peculiar restlessness, occasionally frightful convulsions; the eyes are bright and glistening, but at times suffused; thirst and aversion to fluids characteristic, even the sight or noise of fluids induces paroxysms, with frequent and viscid discharge of saliva.	Drawing up of the nose, wrinkling up of the forehead, angles of the mouth drawn towards the cheek-bones, presenting a frightful risus sardonicus. There is an expression of pain, but the eyes are natural; no great thirst, and in general no great aversion to fluids administered in small quantities; rarely any discharge of saliva.

GLANDERS AND FARCY.

GLANDERS.

Lat. *Equinia.* Fr. *Morve.* Ger. *Rotz.*

DEFINITION.—An inflammatory affection of the nasal mucous membrane, produced by the contagion of matter from a glandered horse. In horses the disease is frightfully contagious and incurable.

FARCY.

Lat. *Farciminum.* Fr. *Farcin.* Ger. *Wurm.*

DEFINITION.—An inflammatory affection of the skin and of the absorbent system, produced by the contagion of matter from a horse having glanders or farcy.

Glanders commences with malaise and general feverish symptoms, and violent pains in the joints and muscles. There is an erysipelatous redness of the face and eyelids, together with a characteristic pustular eruption on the face, sometimes also present on other parts of the body. The eruption is first papular, then vesicular, and at last passes into bullæ or pustules, varying in size from a pea to a sixpenny piece. It may at first be mistaken for the papules of small-pox, the vesicles of chicken-pox, and lastly, the bullæ of pemphigus. When the pustules burst a thin sanious purulent fluid runs from them. The eruption generally begins in twenty-four hours after the general symptoms have commenced. The characteristic "farcy buds" may occur at any period of the disease. The discharge from the nose, also characteristic of this disease, is a thin sanious acrid fluid, becoming thicker as the disease progresses. As death approaches, the symptoms increase in severity, and assume a typhoid type.

In farcy, which is only a variety of glanders, the poison seems to gain entrance into the system through a wound which becomes inflamed, painful, and ultimately gangrenous. Febrile symptoms, not unlike those in glanders, also occur during the course of the disease, but the nasal discharge is most frequently absent

The production of subcutaneous lumps—farcy buds—are characteristic of this disease. These become inflamed and form small abscesses, which, bursting, become unhealthy sores. The lymphatic glands may also become swollen, and suppurate. The patient, worn out with suffering and consequent exhaustion, dies. Glanders and farcy may both occur, either in an acute or in a chronic form. The chronic form is rare. After death, the internal organs present all the appearances seen in severe cases of pyæmia.

Do not confound these diseases with syphilitic coryza or scrofula.

Treatment.—Mercury has been tried, but with little success. Iodide of potassium may be given; and inhalation of creosote is useful as an application to the inflamed nasal passages. Injections of carbolic acid into the nose may be tried. Sulphur used externally, in the form of a sulphur bath, or sulphur given internally, has been recommended by Tardieu. As a rule, however, the disease is fatal in spite of treatment. When the disease occurs in the horse, the animal must be shot. The stables should be disinfected, and not again used for a year or more.

SPLENIC FEVER.

CHARBON, ANTHRAX, MALIGNANT PUSTULE, WOOL-SORTER'S DISEASE.

Lat. *Febris splenica.* Fr. *Charbon.* Ger. *Melzbrandfieber.*

DEFINITION.—A very fatal disease, attacking cattle and other herbivorous animals, often occurring epidemically, probably belonging to the miasmatic contagious class of diseases and communicable to man. In the lower animals the spleen is greatly enlarged, three to five times its normal size, softened, and of a black colour. An important factor in the disease is the presence of the *bacillus anthracis* in the blood and other parts of the animals attacked.

IN THE LOWER ANIMALS.—Splenic fever, when it attacks the lower animals, occurs under three different forms. In some cases the affected animal is convulsed, the breathing is rapid, insensibility rapidly supervenes, and death takes place at longest in a few hours. In other cases fever of an intermittent type is present with spasms of the limbs, and a discharge of blood with the fæcis. In yet another class we have swellings under the skin of the neck, chest, etc., which may lead to sloughing sores, and to which the term "*anthrax*," from the visible manifestations of the disease, is most properly applied. The term "anthrax" is an unfortunate one, as it is the well known classical equivalent for carbuncle. Cattle fed on low marshy land are those most frequently attacked; and it has been suggested that worms feeding on animals buried in the ground, on coming to the surface, deposit on the grass the germs of the specific bacillus, which are thus swallowed by the grazing animals and again give rise to a fresh epizoötic. At any rate it is not wise to pasture animals on land where animals dead of the disease have been buried. The disease, as a rule, prevails most during the hot months of the year, and is most frequently met with in Poland, Hungary, and some of the Danubian Provinces. It is far from unknown in Siberia, and occurs among the wild, as well as among domesticated, animals.

IN MAN.—In man the most common variety of the disease, and until recently the only recognised form, was malignant pustule or charbon. The symptoms of this affection during the first twenty-four or forty-eight hours are characterised by the presence of a small itching spot, most frequently on the face, accompanied with the presence of a small serous vesicle. During the next period, under the vesicle an indurated base is formed of a dark colour, and intensely itchy. The skin becomes red, and there is considerable engorgement of the part and a rapid extension of the process of sloughing. General symptoms of an adynamic type now make their appearance, and unless the disease be early arrested a fatal termination is, as a rule, the result.

The treatment consists in an early destruction of the part with the chloride of zinc, and the administration of tonics, and a supporting diet.

The disease may appear in two other forms, as a "gastro-intestinal" affection, or attacking the thoracic viscera, the now well known "wool-sorter's disease." In the first variety, there may be, together with the gastro-intestinal affection, the local presence of malignant pustule. In this affection the patient first complains of malaise, loss of appetite, headache, giddiness, and pains in the limbs. Then follow vomiting and diarrhœa, accompanied with bloody stools and pain in the abdomen. The fever is not severe, but the patient dies in a state of extreme collapse, often preceded by epileptiform attacks. After death the spleen is found softened and pulpy, and the gastric and intestinal mucous membrane studded with discoloured swellings varying considerably in size. The intestinal glands are also enlarged. In the *thoracic variety* intense prostration and laboured breathing are among the earliest symptoms of the disease. Sometimes there may be bloody expectoration, but, as a rule, the auscultatory signs are not well marked. The temperature varies, being sometimes below the normal, in others varying from 102° to 103° F. There may be nausea and vomiting, but seldom diarrhœa. The appetite is not, as a rule, impaired. Death may be preceded by convulsions or coma, but in some cases the mind is not affected. The fatal termination varies from seventeen hours to four or five days. The *post-mortem* appearances are not well marked, for the lungs may be congested or quite normal. The spleen is seldom, if ever, softened. Hæmorrhages may occur in the pericardium and on other parts of the thoracic viscera. The disease must be treated on general principles.

Etiology.—The disease may be caused by eating the badly cooked flesh of infected animals, by inoculation, flies carrying the virus to abraided parts of the human body. But by far the most important factor in the dissemination of the disease is the infection brought by dried skins or the hair of diseased animals. Strange to say, the skins and hair do not seem to produce the disease in those that pack them, but only in those who unpack them, leading to the supposition that the poison may be generated in transit. Washing or wetting the bags and hair seems to render the work of sorting nearly free from danger. Russian horse-hair and a material known as "Van mohair" appear to be the most important carriers of the infective material of the disease.

GROUP *B.*

MISCELLANEOUS DISEASES.

The affections placed in this Group often invade different parts of the same body, either simultaneously or in succession. They also present certain hereditary tendencies.

RHEUMATIC FEVER. ACUTE RHEUMATISM.

Lat. *Rheumatismus acutus.* Fr. *Rheumatisme articulaire aigu.* Ger. *Acuter Gelenkrheumatismus.*

Definition.—A specific febrile disorder, characterised by non-suppurative inflammation of the fibrous tissues surrounding the joints, of which many are affected at the same time, or in succession.

Acute rheumatism is held by some to be essentially a blood disease, the poison being supposed to be either lactic or some other acid. Dr Bristowe, however, maintains that it is not a blood disease any more than pneumonia, and that "no excess, however, of lactic or any such acid has as yet been detected in the blood or perspiration of rheumatic patients, and if there be a rheumatic poison, which is possible, its discovery is in the future."

Symptoms.—The primary symptoms of the disease are those of a severe catarrh, attended with considerable fever, rigors, and pains in the back and limbs. After a few days, the local attack begins as an acute inflammation of the fibrous structure of one or more of the large joints. The ligaments, aponeuroses, fasciæ, and tendons surrounding the wrists, shoulders, knees, and ankles are most frequently attacked. The affected joints—chiefly the larger ones—are swollen, hot, slightly reddened, and extremely painful, the pain being increased by the slightest attempt at motion. The affection of the joints seldom ends in suppuration or permanent disorganisation of the parts affected. During the course of the attack subcutaneous fibrous nodules, varying in size from a pin's head to a filbert or larger, have been noticed in the neighbourhood of the joints, especially behind the elbows, patellæ, etc. They occur, as a rule, among children. Dr Barlow, who has noticed that they are associated with heart disease, considers that they are connected in character with the exudations forming the basis of valvular disease. A visit made in the evening may find the patient with a painful swollen knee, in the morning his ankle is affected and the knee comparatively well. There is little or no sleep, and sometimes delirium, but, as a rule, head symptoms are absent in rheumatic fever. When they are accompanied with a very high temperature—108° to 110° F.—and a dry skin, a fatal termination may be feared. The fever is peculiar; the skin, though hot—100·4° to 104° F.—is bathed in a profuse, sour, offensive-smelling, and clammy sweat. The urine is very acid and high coloured, and deposits a reddish or pink sediment of urates. The pulse varies from 90 to 110, and is remarkably round and full. The tongue is covered with a brownish fur, and there is entire loss of appetite and obstinate constipation. Any great increase of temperature betokens heart complications. A relapse may take place after an attempt at convalescence, all the symptoms returning with double force. The proportion of the fibrine in the blood is greatly increased, and the blood, when drawn from a vein, after standing for a short time, is *cupped* and *buffed.* Fibrinous coagula found in the heart after death also point to excess of fibrine in that fluid. The tendency to metastasis is great in rheumatic fever, the local inflammation moving from one joint to another, or to other fibrous structures, as the *pericardium, valves of the heart*, the fascia lining the thoracic cavities and diaphragm, and the aponeuroses of the abdominal muscles. The duration of the disease is usually three weeks; but it may continue longer, and become a chronic affection.

Several varieties of rheumatism have been described by authors; thus we have—

Sub-acute Rheumatism.

Gonorrhœal Rheumatism.—Rheumatism associated with gonorrhœa. Generally occurring as a subacute affection of the knee and other large joints. The disease appears to have some obscure relationship to pyæmia.

Synovial Rheumatism.—A rheumatic affection, in which an accumulation of non-purulent fluid occurs in the synovial sacs, and especially in those of the knee joints.

Muscular Rheumatism.—Pain in the muscular structures increased by motion—

(*a*) Lumbago. (*b*) Stiff neck.

The so-called "growing pains" in the muscles of the legs are of rheumatic origin.

Chronic Rheumatism.—Chronic pain, stiffness and swelling of various joints.

Chronic osteo-arthritis. Syn.—*Chronic rheumatic arthritis. Arthritis deformans — nodular rheumatism. Rheumatoid arthritis.* — An affection characterised by pain, stiffness, and deformity of one or more of the joints, associated with deposition of new bone around them. The cartilages of the joints, the starting-point of the disease, become nodular and fringed with fibrous and cartilaginous growths, which early become ossified, and thus form rounded irregular osteophytes like the gutterings of a wax candle. The ends of the bones also become eburnated and highly polished like ivory or porcelain. Suppuration in the joints is rare. In time, the joints become most curiously distorted; the fingers, when the hand is attacked, having the appearance of having been tied up in a knot. Rheumatoid arthritis is essentially a disease of middle age. The knee-joint being most frequently affected in the female, the hip in the male.

The rheumatic pains in scarlet fever are somewhat analogous to gonorrhœal rheumatism, and own a pyæmic source, especially when the sore throat is severe and there is much loss of tissue. The connection between dysentery and rheumatism as a sequel of the former, is well marked, and may be due to pyæmia, the joints being attacked, and sometimes the heart. At any rate, epidemics of dysentery have been followed by rheumatism closely following the bowel disorder.

Causes.—Among the predisposing causes of acute rheumatism, the most important are hereditary influence, adolescence, previous attacks, the male sex, moist and temperate climates and seasons, and a debilitated condition of the system. The exciting cause is usually some sudden chill, induced by exposure to wet and cold, sitting in wet clothes, or in a draught, etc.

Complications.—These form a most serious characteristic of this disease. The organs most affected are the heart: endocarditis, pericarditis, and valvular diseases being common sequelæ of acute rheumatism; the lungs, resulting in pneumonia, bronchitis, and pleurisy; rarely the peritoneum and cerebral and spinal meninges. The eye frequently suffers.

The most important complication in acute rheumatism is when the heart becomes secondarily affected. The affection of the heart should be looked for on the second or third day of the fever; sometimes the heart affection may even precede that of the joints. The symptoms which betoken the heart complication are attacks of dyspnœa, palpitation, and pain increased by any attempt to lie on the left side. Chorea is also a well-marked complication of this disease, and strange to say, the greater number of choreic patients have had an attack of rheumatism and some amount of heart affection as well. An attack of rheumatic fever may lead to the following chain of diseases:— 1. Rheumatism. 2. Endocarditis and valvular lesion. 3. Clot from heart plugging the middle cerebral artery. 4. Softening of the part of the brain supplied by the artery. 5. Aphasia.

Diagnosis and Prognosis.—For the diagnosis see "Gout." The prognosis is favourable if metastasis has not taken place to the brain, heart, or lungs, and if these complications be not very severe.

Treatment.—In the acute form, the treatment consists in the administration of a brisk saline purge in the early stage of the disease, followed by the exhibition of the salts of potash, combined with the iodide of potash and colchicum. The painful joints should be wrapped up in cotton, wool, or flannel. The thirst may be allayed by lemonade or barley water. The diet should be light and simple. The pain may be relieved by opiates—compound ipecacuanha powder at bedtime, and by the application of anodyne fomentations to the painful parts. Of late salicylic acid has been used with most beneficial effect in the treatment of this disease. The following formula may be used:—℞ Acid salicylic, ʒi; liq. ammon. acet, ℥ii; aquam chloroformi, ad ℥vi. Mix. Sumat, ℥i quârtaquâque horâ. The salicylate of soda is now most used. It may be given in fifteen to thirty grain doses every two hours. Its action must be carefully watched, and the periods of administration increased, or the dose lessened if necessary. Under its action the temperature soon falls and the pain becomes less acute; the size of the dose may now be lessened, or it may be given less frequently. Salacin is used by many in preference to the salicylates.

A patient with a temperature of 104° F. will be found to cool down to 99° or lower, after the administration of a few doses of the acid or soda salt. The benefit derived from the use of the acid or its salt is most marked, and in adults it should always be tried. The daily use of the potash water as a drink often affords relief. In chronic rheumatism, the iodide of potash, guaiacum, and colchicum, together with the bicarbonate of potash, are the remedies of most service. In chronic rheumatism, where the pain is increased by the warmth of the bed, the iodide of potash and guaiacum mixture are the best remedies, with the outward application of equal parts of chloroform and aconite liniments. Lemon juice, in tablespoonful doses, has been recommended by Sir G. Burrows and others in acute rheumatism. After the acute stage, small blisters applied to the joints do good. These should be placed on the proximal side of the affected joint, in the form of a circle. The treatment of rheumatoid arthritis is not very satisfactory; that given for the chronic rheumatism may be tried, especially the iodide of potassium.

The heart complication is best treated by dry cupping, and the application of belladonna and mercurial plasters and small blisters to the cardiac region of the chest. (See "Pericarditis.")

GOUT, OR PODAGRA.

Lat. *Podagra.* Fr. *Goutte.* Ger. *Gicht.*

1. Acute Gout.

Definition.—A specific febrile disorder, characterised by non-suppurative inflammation, with considerable redness of certain joints, chiefly of the hands and feet, and, especially in the first attack, of the great toe, and attended with excess of uric acid in the blood.

2. Chronic Gout.

Definition. — A persistent constitutional affection, characterised by stiffness and swelling of various joints, with deposits, brought on by improper food, of urate of soda.

Pathology.—Gout is a blood disease attended with symptomatic fever, the presence of large quantities of uric acid in the blood, and deposits of urate of soda in the immediate neighbourhood of one or more of the small joints, especially the metatarso-phalangeal joint of the great toe. The disease may be the result of some disordered action of the liver producing lithæmia (*q.v.*), or to disturbed action of the kidneys preventing free excretion of uric acid. The normal daily excretion of uric acid by the kidneys is from eight to ten grains, in acute gout it may sink to less than four grains. The deposition of urate of soda in the tissues appears to be in relation to their normal vascularity, those least vascular being attacked. The election of the metatarso-phalangeal joint may be due to the pressure of the weight of the body and its liability to sudden shocks. The kidneys sometimes become affected, the "gouty kidney" of Todd consisting in a diminution in the size of the organ, the capsule being thickened and opaque, and the surface granular.

Etiology.—Gout is more common among men than women, occurring chiefly in elderly men addicted to the pleasures of the table. It is also more common among beer drinkers than among those who indulge in ardent spirits. It is often hereditary, especially in those who suffer habitually from heartburn, and other symptoms of acid dyspepsia, accompanied with copious deposits of uric acid gravel in the urine.

Symptoms.—An attack of gout generally comes on during the night as an acute inflammation of the synovial membrane of one of the smaller joints. The individual in ordinary health, or flattering himself that he is unusually well, goes to bed at his accustomed time; after a few hours' sleep (generally from one to four o'clock in the morning), and generally without any premonitory symptoms, he awakes with pain, more or less intense, in the ball of one great toe, accompanied with shivering. The pain increases—is burning and throbbing; the joint is tense and stiff, and more or less febrile disturbance is also present. After a few hours the symptoms abate, and the patient sleeps a little, a gentle perspiration ensuing on his awakening. Sometimes the attack is ushered in by well-marked premonitory symptoms, which, after a first attack, the patient learns to appreciate. These generally take the form of dyspepsia, with palpitation of the heart, and an intermittent pulse. Troublesome dyspnœa is often relieved by an outburst of apparently pent-up gout. The joint in the morning is found swollen and exquisitely painful: the skin is red, hot, tense, and shining. The veins from the toe are apparently inflamed, distended, and full of blood, and there is slight lividity. The symptoms, less severe in the morning, increase towards night, which is usually spent in renewed restlessness and suffering, again abating as day dawns, Sometimes the pain ceases suddenly, to return at night. During the height of the inflammation the urine is scanty, high-coloured, clear, or deposits a dark-reddish sediment on cooling. During the paroxysm the appetite is impaired, the thirst great, and the bowels constipated. The pulse is not quickened unless the pain be very great. The condition of the tongue will depend upon the state of the stomach and bowels, which are generally deranged. When the fit is about to terminate, the inflamed part becomes gradually less tense and swollen; pitting is more readily produced. The enlargement of the blood-vessels disappears, and after a few days, itching of the skin and desquamation ensue, the joints remaining tender for a short time. After repeated attacks the joints may become greatly deformed. The fingers frequently show the deformity of the joint, to a most marked extent. Metastasis from one joint to another is not unfrequent, and if the patient is not careful, and exposes himself to cold, the internal organs may be attacked, which result is known as *retrocedent gout.* The character of the inflammation of the joint is peculiar; it is very intense, but differs from that of an injury, and is not followed by suppuration. The œdema of a gouty part, and subsequent desquamation of the cuticle, is a peculiarity of gout when superficial structures are attacked. During the early stage of an attack, œdema cannot be observed—that is, when the inflammation is violent and the skin distended and shining; but when the inflammation subsides pitting is easily produced, and the presence of fluid evident. As a rule, true rheumatic inflammation is not accompanied with œdema; when it is, it is not confined to inflamed parts, but affects the whole limb. The profuse sweating in rheumatism is absent in gout. The uric acid of the urine almost disappears, and can readily be detected in the serum of the blood. One attack of gout is usually followed by a second and third seizure, till the disease eventually assumes a chronic form. Hiccough, often troublesome to the patient, not unfrequently occurs in those who have a hereditary tendency to gout. The deposits in the joints known as "chalk-stones," or tophi, largely composed of urate of soda with a little lime, often lead to anchylosis of the joints, and sometimes, by a process of atrophy and ulceration, reach the surface and become exposed. Suppuration is very rare.

Prognosis.—The prognosis in most cases is favourable; the immediate danger to life is slight, but there cannot be a doubt that it tends to shorten life, and Insurance Companies require increased premiums for the "risk."

Diagnosis.—From rheumatism.

1. The deposit of urate of soda in the joints *does not occur* in rheumatism. These concretions, however, are more frequent in chronic than in acute gout.
2. The *small joints* of the hands and feet are the seat of the primary local affection in gout; in rheumatism, the *large joints* of the limbs are affected. It is highly probable that the deposit of urate of soda *precedes* the inflammatory action in the point as cause and effect. In gout, deposits may occur on the helix of the external ear. Heart complications are also rare in gout.
3. The blood in gout is abnormally charged with *uric acid;* in rheumatism *lactic acid* is said to be present.
4. Rheumatism attacks the young; gout seldom occurs before puberty, and is oftener hereditary than rheumatism.

To test for uric acid in the blood, take two drachms of the serum, or the fluid raised by a blister applied at some distance from the inflamed joint, and place them in a large watch glass, and then acidulate with acetic acid. Place a piece of thread in the prepared fluid, and let it remain for some time. Examine the thread with a microscope for crystals of uric acid, and confirm by adding nitric acid and ammonia to form the purple colour murexide.

Treatment.—At the commencement of an attack, the patient should be placed in bed, and the affected limb raised on pillows. A brisk purgative should be given—the sulphate and carbonate of magnesia, combined with colchicum wine, will answer the purpose. My plan of treatment in the acute attack is to give a dose or two of the sulphate and carbonate of magnesia, and, as soon as the bowels are freely relieved, to continue the mixture in smaller doses, but with the addition of 10 or 15 minims of colchicum wine. If the stomach is loaded, an emetic is of great use; but, as a rule, patients don't like to be made to vomit. Dr Garrod recommends colchicum in ʒss and ʒi doses at first, followed by doses of ♏x or less. The painful joint may be surrounded by flannels wrung out in hot water, in which some common washing soda has been dissolved, and then sprinkled with equal parts of laudanum and colchicum wine. Cold applications should be strictly avoided. Mercurials should not be given in gout, as they are not well borne and may do harm. To procure rest, ten grains of the compound ipecacuanha powder may be given at bed-time. If the vital powers are low, omit the colchicum, and give the sesqui-carbonate of ammonia. In chronic gout, the bicarbonates of potash or lithia should be given with colchicum, and the removal, if possible, of all the causes which tend to produce or promote the gouty diathesis. In the early stages of an acute attack the diet should be restricted; animal food either entirely excluded or considerably lessened, and a vegetable diet enjoined. Of course in debilitated patients a restricted diet is not applicable. During the intervals between the attacks, measures should be taken to correct any irregularities in the mode of life; and tonics may be given if necessary. Should the stomach be attacked, hot cordials should be given and the feet put in hot water, with mustard plasters to the stomach.

DIFFERENTIAL DIAGNOSIS OF GOUT, RHEUMATISM, AND RHEUMATOID ARTHRITIS.

GOUT.	RHEUMATISM.	RHEUMATOID ARTHRITIS.
1. Strongly hereditary.	1. Less so than gout.	1. Less so than in gout.
2. Much more frequent in males.	2. As frequent in females.	2. More frequent in females.
3. Seldom occurs before puberty, generally much later.	3. More frequent in the young before middle age.	3. Occurs in both young and old.
4. Induced by high living, wine, and malt liquors.	4. Occurs in the weak, and not caused by wine, etc., excited by cold and damp.	4. Often induced by depressing causes, and sometimes excited by cold.
5. One or more of smaller joints particularly affected in early attacks, and especially great toe.	5. Large joints more affected than small, usually several in number.	5. Large and small about equally affected.
6. Great pain, œdema, and desquamation of cuticle.	6. Pain less intense, seldom œdema.	6. Less pain, much swelling, and often some œdema.
7. Does not induce acute inflammation of structures of the heart.	7. Often causes acute pericarditis and endocarditis.	7. No tendency to cause disease of the heart.
8. Febrile disturbance moderate.	8. Febrile disturbance great, more than from local inflammation.	8. Little febrile disturbance.

GOUT.	RHEUMATISM.	RHEUMATOID ARTHRITIS.
9. Paroxysm periodic in early attack.	9. Attacks not periodic.	9. No periodicity.
10. Early attack lasting a week or ten days.	10. Attacks generally much longer.	10. Duration of attack indefinite.
11. Blood rich in uric acid.	11. No uric acid in blood.	11. No uric acid in blood.
12. Constant deposit of urate of soda in inflamed cartilage and ligaments.	12. No deposit of urate of soda.	12. No deposit of urate of soda, or ulceration of cartilage.
13. Often leads to kidney disease.	13. No tendency to cause kidney disease.	13. No tendency to cause kidney disease.
14. Often produces chalk stones externally.	14. Never causes chalk stones.	14. No chalk stones produced, but swelling of the joints. —*From* GARROD.

DIABETES MELLITUS.

Lat. *Diabetes; Diabetes mellitus.* Fr. *Diabète; Diabète sucré.*
Ger. *Diabetes; Zuckerharnruhr.*

DEFINITION.—A disease characterised by an increased discharge of pale urine of high specific gravity, and persistently impregnated with sugar, accompanied with great emaciation, and sometimes blindness.

Diabetes mellitus, or saccharine diabetes, is a cachectic disease characterised by a profuse flow of pale straw-coloured urine loaded with sugar.

Symptoms.—The first symptoms that may attract attention are an intense thirst and an increase in the flow of urine; but in some cases the patient may complain of having felt a sense of weakness for some time, and which he states is daily increasing.

The specific gravity of the urine is high, being about 1030 to 1040, or more. The colour of the urine is pale, almost colourless in some cases; not, however, due to diminished discharge of colouring matter but to excessive dilution. The quantity secreted in twenty-four hours has been as much as thirty pints. The increase in the urinary flow is most probably due to the irritation of the sugar, causing increased activity of the secreting cells of the kidney. The urea is increased, due to the increase of sugar causing increased decomposition of albuminous tissues. The excess of urea may also be due to the large amount of food taken by the patient. Over two pounds of sugar may be given off daily by the patient *(Stirling)*.

Besides the above-mentioned abnormal flow of urine, the patient gradually emaciates and becomes extremely debilitated. The skin is dry and rough, and the tongue is also dry, red, and fissured. The thirst is intense, and the appetite frequently voracious. Digestion is more or less impaired, and pain and flatulence are complained of after meals. Constipation is the rule, sugar being also present in the fæces. The liquor amnii, in one case I examined, containing as much as 5·6 per cent. of sugar. The breath has a sweetish odour, acetonæmia, not unlike new-mown hay; the urine also partakes of the same smell. The gait is unsteady, the patient walking with an uncertain step. The gums are softened, at times reddened, at others pale, and have a great tendency to bleed on the slightest pressure. The teeth become carious, due to the amount of acid saliva secreted. The pulse is seldom affected; if at all, it is oftener retarded than quickened.

Diabetics often suffer from eczema and other diseases of the skin, and not unfrequently from boils and carbuncles. In all cases of severe *pruritus vulvæ*, accompanied with a dry lichenous or eczematous eruption, it is always well to examine the urine for sugar.

Impaired vision, due in some cases to amaurosis, impairment of the power of the ciliary muscle, to atrophy of the optic discs, and in others to the presence of cataract, due to turbidity of the lens from the amount of sugar in the fluid of the eye extracting water from the lens *(Kunde Heubel)*, is also not an uncommon complication of diabetes.

The course of the disease is not generally very rapid; but it must, nevertheless, be borne in mind that as soon as the glycosuria becomes permanent, the days of the patient are numbered. The prognosis is always grave. Coma may precede death, " but neither acetone nor its precursor, aceto-acetic acid, nor æthyl-diacelic acid, nor the unknown substance in diabetic urine which gives the red colour with ferric chloride *(U. Jaksch)* is the cause of the coma " *(Frerichs and Breiger)*.

Etiology.—Age seems somewhat to influence the occurrence of diabetes; it is rare in infancy, youth, and advanced life, generally recurring in those between thirty and fifty years of age. Mental emotion, anxiety, injuries to the head, over-mental exertion, etc., have all been assigned as causes of diabetes. The presence of syphilitic gumma in or about the floor of the fourth ventricle is held to be one of the causes of the disease *(Dickinson)*.

Pathology.—The pathology of this disease is obscure. By some it is considered to be brought about by impairment in the digestive organs; by others, to be due to hyper-secretion of sugar by the liver, dependent on morbid excitation of the nervous system. The liver, as one of its chief functions, forms a substance known as glycogen, from the sugar and peptones supplied to it from the intestines. In this organ it is stored up, and when required transformed by a diastatic ferment in the liver into grape sugar, which is then carried by the blood into the general circulation, where it is used up. If the circulation of blood through the liver, or the supply of blood to it be increased, temporary glycosuria may follow. This may be due to the accumulation of blood in the liver, thus giving more time for the blood ferment to act on the glycogen and transform it into sugar. Puncturing the floor of the fourth ventricle will cause sugar to appear in the urine. It should be noted that when glycosuria is present, it "may be set aside by section of the splanchnic nerves;" probably by causing anæmia of the liver. "We cannot say whether the temporary diabetes is a simple effect of dilation of the hepatic arteries which accompanies the diabetic puncture, or of some direct action of the nerves on the metabolic activity of the hepatic protoplasm" *(Foster).* Eckhard holds that diabetes produced by puncture of the brain to be due to irritation; Lyon and Aladoff believe it to be the result of simple loss of vascular tone. An excessive saccharine diet, or the inhalation of ether or chloroform, has also been known to produce a like effect. The muscles of the body appear also able to transform sugar into glycogen, and glycogen again into sugar, and the sugar thus formed into lactic acid and glycerine, which undergoes combustion in the blood, and maintains the animal heat. Pavy maintains that "the liver is essentially a sugar-assimilating, instead of a sugar-forming organ, and that when its assimilative action is properly exerted, so little sugar is allowed to pass into the general circulation that the quantity existing in arterial blood is insufficient to render the urine more apparently saccharine than is observed in the healthy state; but that when its assimilative action is not properly exerted sugar is allowed to pass, and in proportion as it does so the urine acquires a more or less marked character." Dr H. Fagge held that diabetes is due to an excessive flow of sugar from the liver into the blood; that the saccharine or amylaceous articles of food exert some directly injurious effect on the liver, resulting in an *excessive production of glycogen* itself by the liver. These are some of the theories put forward to explain the causation of diabetes, but they await the results of future investigation. In the majority of cases the liver is not found diseased, although sometimes smaller than usual. The kidneys are large, and are not unlike in appearance to the large white kidneys of Bright's disease. The lungs are nearly always diseased, phthisis being the most common affection. The brain and alimentary canal are as a rule healthy.

In forming a **Diagnosis**, it must be borne in mind that sugar may appear in the urine in the course of many diseases. It is the persistent occurrence of this substance in abnormal quantities which mark diabetes or glucosuria.

The following are the tests employed for the detection of sugar in the urine :—

1. *Trommer's Test.*—To the suspected urine in a test tube, a small quantity of solution of sulphate of copper is added, enough to make it *slightly* greenish, and then an excess of liquor potassæ, enough to re-dissolve the precipitate that is at first thrown down. If the liquid be now heated to boiling point, and sugar be present, a reddish-brown precipitate of sub-oxide of copper will appear, owing to the carbonaceous nature of the sugar depriving the copper salt of a portion of its oxygen. *Fehling's Solution* is composed of a mixture of sulphate of copper, tartrate of potash, and caustic soda, and may be used instead of the above solutions. It gives far more delicate results, but should always be *first* boiled in the test tube and the suspected urine *added.* Omitting to take this precaution may lead to serious error, as Fehling's Solution does not keep well, and may thus give the reaction of sugar, in the absence of that substance.

2. *Moore's Test.*—The urine is mixed with half its bulk of liquor potassæ in a test tube, and boiled for four or five minutes; if sugar be present, the whole of the liquid will become of a dark-brown colour, from the sugar becoming *glucic acid,* which combines with the potash.

3. *Fermentation Test.*—A bit of dried German yeast is fixed within the mouth of a test tube, which is then filled with urine, warmed to 70° F., and inserted in a vessel containing a small portion of the urine. Fermentation soon takes place, bubbles of carbonic acid gas rapidly rise and collect in the upper part of the tube. Each cubic inch of gas corresponds to a grain of sugar decomposed.

Roberts' Fermentation Process—

1. Collect the urine for twenty-four hours and carefully measure it.
2. Provide two eight-ounce bottles, and into one put four ounces of urine and a small piece of yeast; into the other, only the same quantity of urine.
3. Cork both bottles, and put aside for twenty-four hours in a warm place.
4. Pour the contents of each bottle into two urine glasses, and take the sp. gr. of both.
5. Rule.—*The difference of each degree lost in the urine which has the yeast indicates the presence of one grain of sugar in every fluid ounce of urine.*
6. Multiply the amount of urine× passed in twenty-four hours by the difference of the sp. gr. between the fermented and unfermented urine. The result is the amount, in grains, of sugar passed in twenty-four hours.

Quantitative estimation of Sugar—

1. Make two solutions as follows:—
   (*a*) Crystallised sulphate of copper, 34·63 grms.

× Number of ounces.

Distilled water, a litre. One c.c. = 0·005 grm. of sugar.

(*b*) Rochelle salt, 173 grms.; hydrate of potassium, 80 grms.; water, a litre. Of those solutions 10 c.c. of each are used together.

2. Collect the urine for twenty-four hours, and measure.
3. Take 10 c.c. of urine, and add 200 c.c. of water, and place the mixture in a graduated burette.
4. Place 50 c.c. of water in a porcelain dish, and add 10 c.c. of each of the above solutions, and boil.
5. Now run into the porcelain dish slowly some of the dilute urine, and stir.
6. As soon as the liquid in the dish is colourless, stop the addition of urine, for the process is complete.
7. Rule.—*Divide the number of c.c. of urine passed in twenty-four hours by the number of c.c. of dilute urine required to decolorise the solution in the dish; the result is the amount of sugar passed in twenty-four hours.*

Treatment.—The treatment, for the most, part consists in a very regulated diet. All starchy or saccharine articles of food must be avoided, so that no vegetables are admissible except spinach, lettuce, and water cresses, and other like greens. Almond cake and bran bread are to be taken instead of bread. Milk, eggs, and animal food form the usual diet of the sufferer from diabetes. To relieve the thirst, French wines may be allowed, and water acidulated with phosphoric acid. Dr Charteris recommends one-twentieth of a grain of the nitrate or hydrochlorate of pilocarpin to be placed on the tongue three times a-day. It produces an increased flow of saliva.

If there be much nocturnal restlessness, opium may be given to procure sleep. Opium has also been found of use otherwise than merely to cause sleep. Codeia has been greatly recommended by Dr Pavy and by others, and may be given in gradually increasing doses till the patient takes five grains three times daily. Arsenic, morphia, cannabis indica, conium, and hydrocyanic acid, may all be tried.

Hot baths may be tried, and warm clothing insisted upon, so as to protect the patient from cold. Open air exercise should be taken in wet or dry weather, and on no pretext ought it to be postponed.

Alkalies sometimes afford relief, but as a rule, the treatment of this disease is more dietetic than medicinal.

ANÆMIA.

Lat. *Anœmia.* Fr. *Anémie.* Ger. *Anæmie.*
Syn. *Blutarmuth.*

DEFINITION.—A morbid state of the blood, in which the quantity of the red corpuscles is greatly diminished.

Anæmia may result from one or more of the following causes:—1. Loss of blood, as in severe hemorrhages during childbirth, etc. 2. Loss of albumen from exhausting discharges. 3. Defective assimilation. 4. Rheumatic fever, etc.

In this affection the hœmoglobin is deficient, and the red cells of the blood, although not altered in shape, are diminished in number and vary much in size. Unlike their behaviour in normal blood they do not form *rouleaux* readily, are viscid, "and apt to *fall out.*" There is no absolute increase in the number of the white cells, although, in relation to the red, they are more numerous. In chlorosis small red cells, *microcytes*, are often found, and these may be nucleated as in the fœtus.

Chlorosis is a form of anæmia frequently attacking females about the age of puberty. This affection is also known as the *green sickness*, from the peculiar greenish white colour of the skin, and often occurs without any assignable organic cause.

A peculiar and invariably fatal form of anæmia is known as "progressive pernicious anæmia," or "essential or idopathic anæmia," and is characterised as far as the blood changes are concerned by a large and progressive diminution in the number of the red blood cells, which are irregular in size, and often nucleated together with the presence of a considerable proportion of microcytes. The hœmoglobin is lessened in quantity. The white cells, however, undergo but slight change. The cause of this affection is unknown, but it is probably a primary disease of the red cells or the tissues that form them. The marrow of the bones of affected persons is said to be redder than normal and to contain many large cells. Pernicious anæmia occurs chiefly in women, and particularly in those who have rapidly borne children. To the ordinary symptoms of anæmia are added palpitation and breathlessness, emaciation, and anasarca of the feet, but seldom marked dropsy. The skin presents a peculiar *lemon* or *primrose tint*, almost suggesting the presence of jaundice. There may be repeated attacks of vomiting and diarrhœa. There is also a tendency to extravasations of blood in different parts of the body, but especially into the retina. Epistaxis and bloody vaginal discharges are not unfrequent. In a fatal case I have just attended, when the hemorrhage from the nose ceased, a bloody discharge came from the vagina. After a time fever supervenes, and the patient dies in from one to six months.

Symptoms.—The force of the circulation is lessened, and patients suffer from extreme pallor of the face, most strikingly seen in the palpebral conjunctiva of the lower lid, coldness of the surface of the body and extremities. The action of the heart is feeble, often irregular. A systolic bruit may frequently be heard at the base of the heart and along the aorta, subclavian and carotid arteries. Over the jugular vein a humming sound, *bruit de diable*, is often heard, due probably to the descent of the attenuated blood. The system suffers in all its parts, the vital functions being but languidly performed. Mental depression is often most marked, and a painful sense of inertia, or indolence, is experienced. Some amount of hysteria is often present. The cause of this condition of the blood is not always apparent, often obscure, frequently a pathological element of some other disease. The following are some of the diseases with which anæmia is not unfrequently connected : — tuberculosis, carcinoma, some morbid affections of the kidneys associated with œdema of the face; most wasting diseases, hæmorrhages, and passive discharges; menorrhagia, and other disorders of menstruation; hæmorrhoids, and diseases attended with a disordered state of the digestive organs and the malassimilation of food.

Some mineral substances, when absorbed into the system, appear to induce this morbid condition; lead and mercury possess this power in a marked degree.

Treatment.—The indications for treatment are to remove the cause if possible. Correct the disordered menstruation in women. Tonics, when necessary, should be given, especially the soluble preparations of iron, F. 26; and a nourishing diet, composed principally of meat, should be recommended. When the tongue is flabby, give the astringent preparations of iron, F. 27; and in some cases vary the preparation from time to time. Treat the constipation by giving aloes and nux vomica combined, in the form of pill F. 9. In anæmic women exhausted by frequent pregnancies give the phosphates of lime. Gentle exercise in the open air, cold baths, and change of air, will greatly assist in restoring the disordered state of the system. The treatment of chlorosis is the same as for anæmia. The preparations of iron do not appear to be of any value in pernicious anæmia. Try to improve the general health. Very small doses of corrosive sublimate may be tried. Arsenic also appears to be of great use. In anæmia, accompanied with marked digestive disorder, I have found the bicarbonate of soda with calumba, given before meals, to increase the secretion of the gastric juice, followed after meals by 15 grains of lactopeptine, combined with 15 grains of saccharated carbonate of iron, do much good.

RICKETS.

Lat. *Rachitis.* Fr. *Rachitisme.* Ger. *Rhachitis.*
Syn. *Englische Krankheit.*

DEFINITION. — A constitutional disease of early childhood, manifested by curvature of the shafts of the long bones and enlargement of their cancellous extremities.

This disease, most common in large cities among the children of the poor, is due to mal-nutrition of the bones, occurring during the period of development. It may be divided into three stages; the first in which no deformity of the bones is apparent; the second marked by deformity; and the third, in which the deformity is rendered permanent, and may require surgical treatment for its rectification. The recovery of the bones has been held to be due to a simple deposit of calcareous salts, but this suggestion is not in accordance with the usual phenomena of ossification, nor is there any histological evidence in its favour. The normal percentage of lime salts in the bones of healthy children varies from 63 to 65 per cent.; in ricketic children the percentage may fall to 53 to 33.

Rickets is common to all countries, and occurs in both sexes about the period of the first dentition. To bad hygienic conditions, coupled with insufficient or improper food, the presence of rickets is to be attributed. In some cases the disease is hereditary. The pathology of the disease is obscure, but it is now generally admitted that a deficient supply or deficient absorption of lime and phosphoric acid is not by itself capable of producing rickets; in fact, instead of being a result of insufficient supply, it appears to be due chiefly to irritation of the osteoplastic tissue, which may be brought about by the administration of phosphorus, if, at the same time, the lime salts be removed from the food. Heitzmann has asserted that the chief irritant of the osteoplastic tissue is lactic acid, and that to this substance, together with decreased supply or assimilation of lime salts, true rickets is due. Farinaceous foods given largely and improperly, furnish the lactic acid; the deficiency of the lime salts being due to the impoverished milk of the mother, the result of prolonged lactation, debility, etc., or to errors in the digestive organs of the child, causing the rapid elimination of these salts by diarrhœa, etc.

Symptoms.—In the early stages of the disease rickets is not easy of diagnosis. The child is ailing, it does not thrive, it suffers from repeated attacks of indigestion, it is fretful and troublesome. A low form of fever is almost always present, and, as a rule, the child is subject to *profuse night sweats.* It often also starts in its sleep from no assignable cause. The child also objects to be touched or moved, due to a general tenderness all over the body. After a time it is noticed that the long bones of its legs become bent—scimitar-shaped—and the joints enlarged. The shape of the chest is altered, becomes "pigeon-breasted" or otherwise deformed. The child is late in walking, or if having commenced to walk, it walks with difficulty, the limbs becoming more bent from supporting the weight of the body. In some cases the fontanelles are late in closing, yet the child may have been a "beautiful baby born." The osseous deformity may become worse, or the child, in most cases, entirely recovers.

Treatment.—First improve and regulate the diet. This will in most cases alone cure the dyspepsia. Correct the acidity of the stomach by lime water given in milk. Cold sea baths, or cold baths in which sea salt is dissolved, are useful. There is much advantage to be gained by giving patients something to mix in the bath: plain water is beneath contempt. Tonics and fresh air will greatly assist the recovery of the little patient.

PURPURA.

Lat. *Purpura.* Fr. *Purpura.* Ger. *Purpura.*

Definition.—A disease not usually attended with fever, characterised by purple spots of effused blood, which are not effaced by pressure, and are of small size, except where they run together in patches.

Purpura is a disease consequent on some obscure morbid changes of the blood and the capillaries. The exact cause of this disease is not known. It would appear, however, to be due to a disintegration of the cell wall of the red corpuscles, and escape of their contents. Extravasation of blood through the capillaries then takes place into the various tissues of the body. Some maintain that the primary morbid condition is in the capillaries and other small vessels. Small dull-red or purple spots make their appearance on the lower part of the body and lower extremities, which do not disappear on pressure.

The disease most frequently occurs in children of both sexes, and may attack those who are healthy looking. The administration of iodide of potassium ("iodic purpura") may give rise to purpura. Do not mistake flea-bites for purpuric spots.

The spots when small are known as *petechiæ,* when large as *vibices* or *ecchymoses.*

Symptoms.—The disease is usually attended with symptoms which point to general disorder of the constitution. The appetite is variable, and the patient complains of a feeling of languor and weariness. Palpitation of the heart and giddiness often occur. The spleen is sometimes greatly enlarged.

It is frequently divided into two varieties—

(*a*) Simple.

(*b*) Hæmorrhagic—the disease when accompanied by hæmorrhage from a mucous surface.

Treatment.—The treatment will consist in attempts to remove the morbid conditions which give rise to the disease. The diet should be nutritious, and easy of digestion. The preparations of iron may be given, and astringent remedies; gallic and tannic acid will, in the hæmorrhagic variety, be indicated. Some have recommended the abstraction of blood from the arm as a rapid mode of curing the disease, whilst others have advised the administration of turpentine. Arsenic should also be tried. Purgatives in most cases do good, and may be freely administered. Some cases get better with very little or no treatment. In a case mentioned in the *Medical Examiner* (1877), the treatment consisted in giving ergotine hypodermically: the quantity injected was one-third of a grain at each injection; four grains were thus given, followed by recovery.

SCORBUTUS, OR SCURVY.

Lat. *Scorbutus.* Fr. *Scorbut.* Ger. *Scorbut.*

Definition.—A chronic disease, characterised by sponginess of the gums, and the occurrence of livid patches under the skin of considerable extent, which are usually harder to the touch than the surrounding tissue.

The blood is the seat of the primary and essential lesions of scurvy. The disease appears to be due to the want of proper ventilation, to the deprivation of fresh vegetable food, and to exposure to cold and wet. Dr Garrod considers that scurvy arises from a deficiency of certain salts in the food, notably those of potash. It is more common at sea, in ill-ventilated and badly-provisioned ships, than on land.

Symptoms.—Patients at first complain of a feeling of general weakness and lassitude, followed by pains in the body and limbs, modified by rest and sleep. The appetite soon fails, the pallor of the surface of the body is well marked, the gums become spongy, with a tendency to bleed spontaneously, or on the application of slight pressure. Livid patches of considerable extent form under the skin, generally harder to the touch than the surrounding tissue. The skin is dry and rough, and sometimes there is considerable œdema of the face and ankles. The slightest exertion produces attacks of dyspnœa and palpitation. An anæmic bruit is sometimes heard on auscultation. Diarrhœa is sometimes present; at others, troublesome constipation. The breath is fetid, the urine high-coloured, and rapidly undergoes decomposition. Impaired vision is not an unfrequent occurrence. Sometimes also a curious affection occurs among those suffering from scurvy known as "hemeralopia," day blindness, or "nyctalopia," night blindness. During the day the patient can see, and at night by the aid of artificial light he may be even able to read, but, if the artificial light be removed, he becomes perfectly blind and cannot see his way even in broad moonlight. No opthalmoscopic changes appear to be present, but the condition appears to be associated with imperfect conditions of nutrition.

The danger to life depends greatly on the extent of the ecchymoses, and the amount of hæmorrhage that takes place from the mucous surfaces. Death may occur from effusion of blood into serous cavities, or into the substance of vital organs. The heart is often found soft and pale, sometimes fatty, the spleen large and soft, and the blood watery. The joints may contain blood. The intestines may show signs of hæmorrhage as in purpura.

Diagnosis.—Scurvy must not be confounded with purpura. 1. Sponginess of the gums, present in scurvy, is absent in purpura. 2. Scurvy is due to improper diet and a want of green succulent vegetables. The cause of purpura is unknown. 3. Purpura comes on suddenly, scurvy slowly. 4. Scurvy can be cured by fresh air and green vegetables, and especially potatoes added to the diet; this treatment is of no use in purpura. 5. Scurvy affects adults, chiefly sailors on long voyages; purpura, children of both sexes. 6. Scurvy is marked by extreme debility, and is rendered worse by venesection. Purpura is often cured by blood-letting and mercurials.

Treatment.—This will consist in the use of vegetables and ripe fruits, notably those of the orange order. Potatoes which keep well should always be stored in vessels preparing for long voyages. Although the potato is not rich in potash salts, it has been found a great preventive to scurvy. Good ventilation is also essential as a means of cure. The medicinal treatment will depend on the condition of the patients. Ferruginous tonics, quinine, chlorate of potash, etc. The hypodecmic injection of atropia has been recommended. A gargle containing chlorate of potash and myrrh, or chlorinated soda, will be found useful.

TUBERCULOSIS.

Lat. *Dyserasia tuberculosa.* Fr. *Tuberculeux.* Ger. *Scrophulose.* Syn. *Scrofule.*

DEFINITION.—A cachetic condition of the system, manifesting itself in the production of certain diseases known as scrofula, pulmonary consumption, tubercular peritonitis or tabes mesenterica, tubercular hydrocephalus. This state has also been indicated by the terms the *tuberculous*, the *scrofulous*, or the *strumous diathesis.*

The tuberculous or scrofulous cachexia is associated with a constant and steady increase of temperature, a general wasting of the body, and the development in one or more organs of the body of a substance known as tubercle. Arthritis of one or more joints is often a first symptom of a general tuberculosis, due probably to an implication of the synovial membranes in the disease. The development of tubercle may take place in any organ or tissue of the body, but the lungs appear to be more frequently affected than other parts; and of the lungs, the apices are the parts most frequently attacked. In children the lymphatic glands, lungs, brain, intestinal mucous membranes, etc., are the parts in which tubercles most frequently occur. In adults the lungs are the usual seat of the deposit. "It is a characteristic of the constitutional disease called 'scrofula,' that all the inflammatory processes run a peculiar course. There is a well-marked tendency to protraction; the infiltration disappears very gradually, or it remains stationary and undergoes regressive metamorphosis of a cheesy character" *(Cornil et Ranvier)*. By *tuberculisation* is understood the local process by which the elements of a part become metamorphosed into tubercle. Some pathologists consider tubercle to be a non-inflammatory exudation or deposit which undergoes a succession of regular changes from *grey* to *yellow* tubercle; others, that it is a retrograde metamorphosis of pre-existing structures. Virchow holds that tubercle, properly speaking, is not an exudation, but that it has a cellular origin, that the cells are derived from the connective tissue, and that they rapidly multiply.

Many observers now maintain that tubercle has an inflammatory origin, and is in fact a growth formed of cells—"giant cells"—with a tendency to rapid degeneration or *caseation* and death. The student must be informed that there is a "tubercle bacillus," but whether an originator of tubercle or a lodger in the material has yet to be shown. At any rate, the most dogmatic advocates of the "tubercle bacillus," admit that "a predisposition on the part of the individual is necessary; without which tubercular disease does not occur."

Buhl some years ago propounded the doctrine that the infective nature of acute tuberculosis was due to the escape of particles of caseous matter from already existing centres, and that it was to the absorption of these particles that the general tuberculous development was due. Of late this view has been somewhat modified, and experiments have shown, "in the first place, that caseation of an inflammatory product is not necessary for it to constitute an infective focus; and, secondly, that the development of the general tuberculosis is not due to anything specific in the substances innoculated, but that the products of various inflammatory processes (for the most part of inflammations of slight intensity) may constitute the infective agents." In some animals—guinea-pigs and rabbits—tubercle may be produced in the lungs by the simple irritation of a seton in the neck.

The following Table may assist the student:—

NATURAL TUBERCULOSIS.	ARTIFICIAL TUBERCULOSIS.
Anatomical Differences. 1. Brain frequently affected. 2. Changes commence in the alveoli.	*Anatomical Differences.* 1. Brain rarely affected. 2. Development of adenoid tissue around the perevascular lymphatics. Alveoli secondarily affected.
Pathological Differences. Caseation not so extensive as in the artificial production.	*Pathological Differences.* Caseation extensive.

Tuberculosis, although an infective disease, presents marked differences from pyæmia. In tuberculosis the lesions produced are limited to small areas, and the irritation is not very severe; in pyæmia, on the other hand, any degree of severity may result, and abscesses in several organs formed. Tuberculosis has also a tendency to become chronic, pyæmia to run an acute and severe course.

There are two varieties of tubercle—*grey*, living or growing tubercle, and *yellow*, or dead tubercle, with probably a tubercular origin, but sometimes merely the dèbris of other cell growth; and Virchow maintains that no mere cheesy or caseous mass should be considered tuberculous unless grey granules were found. Cheesy masses can be formed from thickened pus.

Hence phthisis may have another than tuberculous origin. Yellow tubercle is probably only a fatty degeneration of the grey.

Grey tubercle consists of masses varying in size, composed of small roundish or oval bodies, about the size of millet seeds, embedded in a granular matrix. It is tough, soft, semi-transparent, compressible, and of a pearly-grey colour. The granules are more or less dissolved, and the corpuscles rendered transparent by the action of *acetic acid.* Potash in solution entirely dissolves them. An inability to *progressive development* is characteristic of tubercle.

Yellow tubercle occurs as opaque masses, usually larger than the grey, friable, and having a cheesy, lardaceous consistence. The tendency of tubercle is to undergo retrograde metamorphosis; to become fatty, to dry up, or to become the seat of calcareous deposits.

To chemical analysis tubercle yields animal matter and earthy salts.

The term Scrofula has been given to a constitutional disease resulting either in the deposit of tubercle, or in specific forms of inflammation or ulceration.

HÆMOPHILIA.

Lat. *Hæmophilia.* Fr. *Hémophilie.* Ger. *Bluterkrankheit.*

DEFINITION.—A congenital affection, characterised by a tendency to bleed on the slightest injury.

Hæmophilia occurs most frequently in males. Some families have a tendency to be "bleeders." Epistaxis is frequently present, in fact hæmorrhage from the mucous surfaces of the body is most characteristic of the affection, and such hæmorrhage may, in many cases, be almost uncontrollable. Morbid anatomy throws little light on the nature of the disease. The treatment consists in taking measures to prevent injury and the use of ordinary styptics should hæmorrhage take place.

CARCINOMA.

SCIRRHUS.

Lat. *Scirrhus.* Fr. *Squirrhe.* Ger. *Scirrhus.* Syn. *Faserkrebs.*

DEFINITION.—Cancer, characterised by hardness of the primary tumour, due to its relatively large proportion of stroma, and by a tendency to draw to itself the neighbouring soft structures. When ulcerated, the sore is commonly deep, uneven, and bounded by a thick everted hard edge, most common on the female breasts.

MEDULLARY, ENCEPHALOID.

Lat. *Carcinoma medullosum.* Fr. *Cancer mèdullaire.*
Ger. *Markschwamm.*

DEFINITION.—Cancer, characterised by a smoothly lobed surface, soft irregular consistence, great vascularity, and usually rapid growth and reproduction. When ulcerated, it protrudes in large masses, which bleed copiously.

COLLOID.

Lat. *Morbus collodes.* Fr. *Cancer colloïde.* Ger. *Colloidkrebs.*

DEFINITION.—A new growth, a great part of which is formed of transparent or gelatinous substance.

EPITHELIAL.

Lat. *Carcinoma epitheliosum.* Fr. *Cancer èpithèlial.*
Ger. *Epithelialkrebs.*

DEFINITION.—Cancer, characterised by its occurrence chiefly in parts naturally supplied with epithelium, and by the resemblance of its cells to those of the epithelium.

The carcinomatous cachexia is characterised by the development of tumours in various parts of the body, which present certain peculiarities. They return after extirpation, are attended with progressive destruction of the part affected, have a tendency to infiltrate the adjacent lymphatic glands, and they often prove fatal, not through their local action, but by causing a peculiar marasmus, which ends fatally.

Four well-marked forms of cancer are recognised—Scirrhus, or hard cancer; Medullary, or soft cancer; Colloid, or alveolar cancer; and Epithelial; but to those have been added others, as osteoid, melanotic, etc.

TABLE GIVING THE GENERAL AND MICROSCOPICAL CHARACTERS OF CANCER, AND ITS USUAL SEATS.

	SCIRRHUS.	ENCEPHALOID.	COLLOID.
General Characters.	Hard firm tumour of slow growth, sometimes with a depression in the centre of the growth, with a puckering of the surrounding skin. The centre firm and hard, the circumference softer.	Soft, as its name implies, and of rapid growth. When cut into, the tumour presents a brain-like appearance.	A consistency partaking of a mixture of the two former. Gelatiform appearance when cut into.
Microscopical Characters.	This form of cancer is characterised by the large amount of the stroma. The epithelial portion degenerates and decreases, and the fibroid stroma increases. Large cells of varying forms containing nuclei and nucleoli, but "there is no specific cancer cell;" areolæ bounded by a fibrous structure containing epithelial cells. Characters common to glandular structures, but wanting in regularity of gland structure.	Smaller amount of stroma than in scirrhus. The epithelial portion grows rapidly, and undergoes fatty degeneration at the expense of the fibrous portion.	The alveoli with thin walls and containing gelatinous colloid material.

TABLE—*Continued.*

TABLE—*Continued.*

	SCIRRHUS.	ENCEPHALOID.	COLLOID.
Seat.	Female breast, pylorus, œsophagus, rectum.	Usually as a growth secondary to scirrhus. *Primarily* sometimes in the ends of bones, the eye, testicle, etc.	Stomach, intestines, peritoneum, omentum, etc.
Propagation and Activity.	Propagates itself in other organs more in form of encephaloid than in the primary form.	Rapid propagation due to its vascularity and activity of its growth.	Not so active as scirrhus or encephaloid.

SECTION III.

LOCAL DISEASES.

DISEASES OF THE RESPIRATORY ORGANS.

PRELIMINARY REMARKS.

The student cannot too early learn the necessity for making a careful examination of his patient in a methodical manner, for in no other way can he ensure the correctness of his diagnosis, and be of benefit to his patient. Not as a mere matter of routine we offer him the following scheme, which we shall divide under the following heads:—*Inspection*, *Palpation*, *Percussion*, and *Auscultation;* and under each we shall try to show the nature of the information for which we are in search.

Inspection.—The patient should be either lying on his back, or standing and stripped to the waist. We may then notice the movements of the thorax, whether they are regular and equal on both sides. The state of respiration, either easy or difficult; quick or slow. Ordinary or enforced respiration, the presence or absence of dyspnœa. The apex beat of the heart, etc. The nose and larynx should also be inspected, and the character and tone of the voice noted. An examination of the sputa should also be made.

Note.—Dyspnœa may be present, due to the existence of diabetes, heart disease, Bright's disease, and paralysis of the diaphragm. Cough—a marked symptom in thoracic complaints—may be due to irritation about the pharynx, to wax in the ear, or disease in the internal structure of the organ — cerebral disease. Hysterical cough.

Palpation.—This is performed by placing the hand or hands on the part affected. We may thus judge of the expansive power of the thorax, of the presence or absence of injury (*i.e.*, fractured ribs), the presence, increase, or diminution of vocal fremitus, the presence of fluctuation, vibrations, crepitation, etc.

Percussion.—Percussion may be either *immediate*, consisting in striking the thorax directly with the tips of the fingers, a method now abandoned; or *mediate*, practised by placing a finger or a plexemeter between the thoracic wall and the percussing finger or hammer. The movement of the right percussing hand *must spring from the wrist only*, the forearm remaining motionless. The patient should, if possible, be in a standing or sitting position, as more accurate results are thus obtained. The arms should hang loosely by the sides when percussing the anterior surface of the thorax, raised above the head while the lateral and axillary regions are examined, and crossed over the chest when the back is percussed. Percussion *should commence* at the supra-clavicular region, and proceed symmetrically downwards on each side. It is almost impossible to give any adequate description of the sounds elicited by percussion, the student is therefore advised to neglect no opportunity of studying the character and tone of normal and abnormal sounds under a competent teacher. Besides the sounds elicited on percussion we may determine the amount of resistence offered to the finger, thus giving a valuable clue to the condition of the organs percussed.

Auscultation.—Like percussion, auscultation of the chest may be either *mediate* or *immediate*, according as we listen *with* or *without* the intervention of the stethoscope between the ear and the chest. The knowledge which auscultation affords can only be gained by practice, the student may, however, be referred to the tables in another portion of this volume. Not a moment is lost by the student in making himself acquainted with the different thoracic sounds.

CATARRH.

Lat. *Catarrhus.* Fr. *Catarrhe.* Ger. *Catarrh.*

Common catarrh or "cold in the head," is an inflammatory affection of the mucous membrane of the upper portion of the air-passages. It consists in an hyperæmic state of the vessels of the mucous membrane, attended with swelling, sponginess, and an abnormal secretion. This disease has received various names, indicating the parts affected; thus, when the nasal mucous membrane is attacked, it is called *coryza;* when the bronchi suffer, *bronchitis;* and so on. The susceptibility of individuals varies greatly, even when exposed to like conditions; and it is remarkable that the same agent which will give rise to a coryza in one, will affect the bronchial tubes or the larynx of another. It may be mentioned that cachectic individuals, and those who are badly nourished, or who lead sedentary and effeminate lives, are more predisposed to this affection than those whose mode of living is more active, even though exposed to great variations of temperature.

The exciting causes appear to be either local irritants acting directly on the part, or sudden chilling of the surface, especially when the body is exhausted, and rapidly cooling. Many drunkards appear to suffer from an habitual catarrh, due, doubtless, to the extension of the catarrhal affection of the stomach, induced by the irritating action of the alcohol.

It should be borne in mind by the student, that, whenever the inflammatory process is set up in a part, it is always attended with—1. Pain. 2. Heat. 3. Redness. 4. Swelling. 5. More or less impairment in the functions of the part affected.

The most probable explanation of the mode in which cold acts in the production of inflammation in any of the internal organs—as is the case with the lungs in pneumonia—is to suppose that the effects produced are in some measure due to reflex action. A portion of the surface is suddenly chilled, and the organ situated beneath, or in nervous sympathy with it, becomes congested and inflamed. During health, there is a certain amount of nervous influence exercised by the nerve-centre on both skin and organ. Cold suddenly applied to the skin destroys the equilibrium previously existing, and the consequence is congestion and inflammation of the organ. The action of counter-irritants in checking inflammatory processes may be explained by supposing that they restore the nervous equilibrium. Hence, also, the value of large opiates, and the application of warmth in the first stages of catarrh, pleurisy, etc. These doubtless act by modifying the morbid or excessive action of the nerves on the organ.

The symptoms which usher in an attack of catarrh are too well-known to need description.

Treatment.—In the acute form of the disease the treatment is simple enough, consisting in the administration of diaphoretics and saline aperients, together with the use of hot baths. For an adult, ten grains of the compound ipecacuanha powder at bed-time often give great relief. A short time ago Dr Ferrier proposed the use of a snuff composed of morphia, gum acacia, and bismuth in the treatment of nasal catarrh. If the disease be taken in its earliest stages, the remedy suggested certainly does good. It is only when the affection assumes a chronic form that the treatment becomes more complicated. Tonics, good ventilation, and warm clothing; all muffling up, however, should be carefully avoided. The throat and chest may advantageously be bathed every morning with cold water, and exposure to cold draughts carefully avoided. It should be borne in mind, that though a simple affair in itself, a catarrhal affection should not be neglected, as serious complications often occur which can clearly be traced to "only a cold."

HAY ASTHMA.

Lat. *Asthma ex fœnisicio.* Fr. *Fievre de foin.*
Ger. *Heu-Asthma.*

Definition.—A catarrhal affection induced in some persons by the exhalations from hay, feathers, ipecacuanha, etc.

This disease, known under the names of *hay fever*, *summer catarrh*, or *hay asthma*, may be described as an acute catarrhal affection of the mucous membrane of the nose, larynx, bronchi, and conjunctivæ. It is attended with an increased discharge of mucus from the nose and bronchi, watering of the eyes, and asthmatic paroxysms, which become most distressing to the patient. Hay asthma is induced in some persons by the odour of newly-mown hay; in others by the emanation from powdered ipecacuanha, and feather beds or pillows, as mentioned by Dr Austin Flint. Hay asthma may be attended with all the symptoms of true spasmodic asthma.

Hay fever is truly not identical with *hay asthma*, the former being more of a congestive than a spasmodic disease, and Dr Pirrie has pointed out that the disease is probably a paresis of the nervous centres. This may explain the reason that hay fever yields more readily to tonics, strychnia, etc., than to the usual remedies of an expectorant nature. It would appear, therefore, to be due more to some idiosyncrasy existing in certain individuals than to any specific poison, as all persons are not affected by the same agents.

Treatment.—The treatment of hay asthma consists in the removal of the exciting cause, change of air, a visit to the seaside, and tonics—quinine or iron—when indicated. Locally, a mixture of vaseline and carbolic acid (gr. 10–20 to the ounce) applied to the interior of the nostrils with a brush, has been found very efficacious in arresting the sneezing and coryza. In hay fever, when the headache is severe, hydrobromic acid may be given with most beneficial results. Saline aperients and tonics may also be required in the treatment of this disease.

OZÆNA.

Lat. *Ozœna.* Fr. *Ozène.* Ger. *Ozäna.*

Definition.—Chronic inflammation of the nasal mucous membrane.

Chronic inflammation of the nasal mucous membrane may be caused by repeated attacks of acute catarrh, or it may occur in strumous subjects without any direct assignable cause. A permanent narrowing of the nasal cavities is brought about by the swelling of the mucous membrane, and from the difficulty of breathing thus induced, this disease has been vulgarly called "the snuffles." It is attended with a fetid muco-purulent discharge from the nostrils, which has a tendency to harden into crusts that may be expelled by blowing the nose. Ulceration may occur; but unless the ulcers are low down, no positive diagnosis can be formed, as the odour of the discharge is not pathognomonic of this condition. The swollen state of the mucous membrane may give rise to the idea of polypus. Careful examinations with a speculum and probe should therefore be frequently made, especially as the discharge may be due to the presence of a polypus, and a cure effected by its removal.

Treatment.—In the treatment of this disease, especial care must be paid to any constitutional defect. Tonics, change of air, the exhibition of cod liver oil, washing out the nostrils with a solution of nitrate of silver, sulphate of zinc, or solution of carbolic acid, 1 part to 60. The enlarged mucous membrane may, if necessary, be touched by lunar caustic. For the thorough cleansing of the nasal passages, the post nasal douche should be used.

LARYNGITIS.

Lat. *Laryngitis.* Fr. *Laryngite.* Ger. *Entzündung des Kehlkopfs.*

Definition.—Acute inflammation of the larynx.

Laryngitis is an active inflammation of the mucous membrane of the larynx, sometimes extending into the submucous areolar tissue, and attended with infiltration, which greatly increases the dangerous tendency of this disease.

Œdema of the glottis may be due to attempts at swallowing the concentrated mineral acids or solutions of caustic potash or soda, or, in the case of children, boiling water. Œdema of the glottis may also be met with in Bright's disease.

Follicular pharyngitis, or, perhaps, more correctly, *follicular disease of the pharyngo-laryngeal membrane*, is most frequently met with in singers and clergymen, and is commonly known as "clergyman's sore throat." The follicles and glands of the mucous membrane are the parts chiefly affected. Clergyman's sore throat, in many cases, is merely a nervous affection. It may arise from over-exertion in talking as well as from cold.

Erysipelatous or *suppurative laryngitis* is a most formidable disease. It not unfrequently accompanies erysipelas of the neck. A purulent secretion takes place in all the tissues of the larynx and neighbouring parts. Pus may be found in the cellular tissue, and even in the muscles of the pharynx.

Symptoms.—The early symptoms of laryngitis are those which usher in most inflammatory affections. To these are added symptoms which point out the locality of the affection. The voice becomes hoarse, and as the swelling increases, is entirely lost. Pain is felt over the pomum adami, and the inspirations are long and frequently attended with a wheezing sound. Deglutition is difficult and accompanied with intense pain. A glairy mucus discharge, more or less tinged with blood, is expectorated, accompanied with a harsh brassy cough. If the disease continue unchecked, the difficulty of the breathing increases, and the suffering of the patient becomes intense, and, as the scene closes, he becomes drowsy or delirious. When death occurs, it is due to suffocation, but suffocation does not necessitate a complete choking up of the passage, since death may result either from spasm of the glottis, due to irritation, or from paralysis of the muscles. When there is œdema of the glottis, the severity of the symptoms is increased, inspiration being loud and noisy, whilst expiration is performed with less difficulty. In adults this disease is rare; in children, it is more common. It may occur in connection with scarlet fever, small-pox, and, though seldom, with pharyngitis. It may also be produced by the inhalation of irritating vapours, and often in children by swallowing boiling water.

Diagnosis and Prognosis.—The presence of fever, together with the local pain, distinguishes acute laryngitis from spasmodic affections of the larynx; and the huskiness followed by entire loss of voice, from croup. The prognosis is most unfavourable when infiltration has taken place, the fatal issue rapidly ensuing. It is more fatal to adults than to children. A more favourable opinion may be given if the inflammatory affection subside, and the breathing become easier and less laboured, and attended with profuse expectoration.

Treatment.—The treatment is that applicable to inflammatory affections in general. The local measures to be adopted are, the application of hot poultices or blisters to the neck, the inhalation of steam, the topical application of a solution of nitrate of silver, or chloride of zinc (20 gr. to ℥i), and, as a last resort, the operation of tracheotomy should be performed. The œdema of the glottis should be treated by incision, or by the application of a solution of nitrate of silver, sixty grains to the ounce, and this solution will also be found useful in cases of follicular pharyngitis or dysphonia clericorum. To the local application must be added a tonic treatment, iron, quinine, etc. In erysipelatous laryngitis, a supporting course of treatment must be adopted. A gargle of chlorinated soda will be found most useful in some cases. Ipecacuanha wine as spray should also be tried, especially in relaxation of the vocal cords.

CHRONIC LARYNGITIS.

Lat. *Laryngitis longa.* Fr. *Laryngite chronique.*
Ger. *Entzündung des Kehlkopfs chronische.*

Definition.—Chronic inflammation of the larynx.

This disease may occur independently of a previous acute attack. The varieties of chronic laryngitis are simple, tubercular or *laryngeal phthisis,* and syphilitic.

Symptoms.—Thickening of the mucous membrane takes place, which may be attended with ulceration. The voice becomes hoarse, or entirely lost. The sputa, when ulceration is present, becomes purulent, or sanious and fetid. In most cases, chronic laryngitis is a concomitant of either syphilis or a tubercular affection of the lungs, hence an examination of the chest should be made in all cases of chronic laryngitis. In some cases, the act of deglutition is not attended with pain; in others, the most distressing symptoms supervene on the attempt to swallow.

Diagnosis and Prognosis.—The permanent morbid change in the voice, the cough, the difficulty in the breathing, together with the tenderness felt when the larynx is pressed, renders the diagnosis easy. An examination of the larynx with the laryngoscope will decide as to the nature of the disease. The prognosis will, of course, depend upon the nature of the diseases with which it is combined.

Treatment.—The local treatment will consist in the application of counter-irritation to the outside of the throat, and the internal application of nitrate of silver, chloride of zinc, or sulphate of copper, by means of a sponge attached to a probang. All laryngeal applications should be made by the aid of the laryngoscope, to the exact spot required, and not swabbed indiscriminately about the throat. The strength of the solution used should be from twenty to forty grains to the ounce of water. The general treatment depends upon the presence of debility, syphilis, or phthisis.

TABLE SHOWING THE POINTS OF DISTINCTION BETWEEN CROUP, WHOOPING-COUGH, AND LARYNGISMUS STRIDULUS.

	CROUP.	WHOOPING-COUGH.	LARYNGISMUS STRIDULUS.
Premonitory Symptoms	Slight Fever and other symptoms of a catarrhal attack, lasting two or three days.	Those of a common catarrh, of eight or ten days' duration.	None; absence of fever, etc. etc.
Characteristic Symptoms	Cough accompanied with a peculiar ringing "brassy" sound. Inspiration is prolonged, and the voice is hoarse.	The air is expelled from the lungs by a series of expiratory efforts; at last, when the patient appears to be on the point of suffocation, a long protracted inspiration takes place. The air, rushing through the half-open glottis, gives rise to the peculiar noise or *whoop*. The fit of coughing ends by the discharge of a quantity of mucus, sometimes by vomiting.	The attack is sudden, generally in the night. There is a sudden interruption to the breathing, due to a spasmodic action of the glottis. When the spasm is relieved, the air is drawn in with a peculiar crowing sound, like a cock.
Pathology and Morbid Anatomy	The formation of a false membrane in the trachea, the result of a peculiar form of inflammation.	No morbid appearances characteristic of this disease. It is contagious.	Absence of inflammation and deposition of a false membrane. Due, probably, to some irritation of the nervous system by teething, improper diet, etc.

CROUP.

Lat. *Angina trachealis.* Fr. *Croup.* Ger. *Croup.*
Syn. *Häutige Bräune.*

DEFINITION.—Inflammation of the larynx, accompanied with exudation of coaguable lymph.

Cynanche trachealis, or croup, is an inflammatory affection of the mucous membrane of the larynx attended with the exudation of coaguable lymph, which in time becomes detached by a muco-purulent exudation under it, and is then expectorated in sheets or in the form of tubes. Croup occurs for the most part in children subsequent to the primary dentition, and up to the seventh or tenth year of age. It is more prevalent in damp places, with northerly aspects than in southerly and protected parts. The exciting causes of croup are not always apparent. Males are oftener attacked by this disease than females.

Pathology, etc.—The chief seat of the false membrane is in the larynx, then in the trachea, and lastly, extending into the bronchi. In severe cases all the parts mentioned may be at one and the same time affected, but in all cases the larynx and trachea are always attacked. At the outset of the disease there is an increased vascularity of the mucous membrane, then an exudation of liquor sanguinis and blood corpuscles, together with an increased proliferation of the epithelial elements. The production of the *false membrane* now rapidly follows. This is a fibrinous layer covering the surface of the larynx and trachea, often commencing just below the epiglottis and thence extending to the bifurcation of the trachea. When the false membrane is examined under the microscope, it appears as a finely fibrillated and delicate net-work, homogeneous in character, and inclosing numerous leucocytes and epithelial cells in its meshes.

Symptoms.—The symptoms of the *first stage* of croup are those of an ordinary cold, except in those cases where the disease has caused death in a few hours. During the *second stage*, the cough is accompanied with a peculiar *brassy* ringing sound, like the crowing of a cock. Each inspiration becomes more prolonged, and is attended with a harsh crackling noise. The pulse is frequent and sharp. The disease appears to increase in severity towards night. The desire for drink is great, and the act of swallowing is not usually attended with pain. If the disease has not been checked, the *third stage*, ending in death, ensues. In the early part of this stage the child lies in a listless condition on its mother's or nurse's lap, rolling its eyes and breathing heavily. Suddenly an attack of convulsions occurs, ending in death, or the child may die either in a state of coma, or in a paroxysmal attempt to breathe.

The **Prognosis** should always be very guarded, especially if the disease be attended with any lung complication.

Treatment.—The treatment, to be successful, must be prompt. The patient should be put without delay into a hot bath, and an emetic of antimony and ipecacuanha administered. Confinement to bed is absolutely necessary, and a saline mixture containing small doses of tartar emetic should be exhibited every few hours. Bleeding in any form should not be used. The atmosphere of the room should be kept moist by the introduction of steam. It is as well to surround the bed with a high screen, and to pass steam into the area thus made so that the child may live in a moist warm atmosphere. Some prescribe the application of cold compresses to the throat, and large doses of sulphate of copper till vomiting be produced. Calomel is also lauded by others.

The prophylactic treatment consists in not confining the child to one room, or clothing it too much, but in the careful avoidance of exposure to northerly or north-easterly winds.

The following Table will show the Various Forms of Dyspnœa, and how they may be Distinguished.

CARDIAC.	BRONCHITIC.	ASTHMATIC.	EMPHYSEMATIC.
Intolerance of the slightest exertion or of the recumbent posture. The breathing has a panting and gasping character. The dyspnœa is more a *breathlessness* than a difficulty of breathing. It is due to the enlargement of the heart, aneurismal tumours, etc.	The breath is short, and attended with cough, crepitus, due to diminished size of air tubes, and accumulations of mucus.	The breath is often long drawn, dry, and without cough. There is a peculiar wheezing sound, showing bronchial contraction, due to spasm of the fibres surrounding the air tubes.	Constant, varies but slightly, and there is no wheezing.

Sounds produced by the Passage of Air in Respiration:—

NATURAL.

Tracheal; in the neck, and at top of the sternum.

Bronchial; near the top of the sternum, and between the scapulæ.

Vesicular; heard over most other parts of the chest, and produced by the entrance of air into the alveoli. It is audible only during inspiration.

MORBID.

Bronchial respiration; from condensed lung.

Cavernous; *Amphoric;* in cavities communicating with the bronchi.

Râles & Rhonchi.

- Moist
  - *Mucous;* liquid in bronchi.
  - *Crepitation;* viscid liquid in small tubes and air cells.
  - *Gurgling;* liquid in cavity.
- Dry
  - *Dry crepitation;* in emphysema.
  - *Cavernous ronchus;* in cavity destitute of fluid.
  - *Sibilant and Sonorous rhonchus.* Contraction of bronchi, by swelling of mucous membrane, pressure, or tenacious secretions.

Sounds of the Voice transmitted through the Chest:—

- Healthy.
  - *Laryngophony;* over larynx.
  - *Tracheophony;* over neck and upper part of the sternum.
  - *Bronchophony;* near top of sternum, between scapulæ, etc.
  - *Fremitus, or vocal vibration;* felt by hand in many parts of chest.
- Morbid.
  - *Bronchophony;* exaggerated sound of voice through condensed lung, unattended with articulation.
  - *Ægophony;* the same vibrating through a thin layer of fluid; occurs during pleuritic effusion of small amount.
  - *Pectoriloquy;* the same in the cavity of the lungs, complete transmission of articulated words from walls of the chest into the ear.
  - *Tinkling, etc.;* a changed echo of voice or cough, in a large cavity containing air and liquid, chiefly in phthisis.

Sounds produced by the Motion of the Lungs:—

Friction Sounds, when the pleuræ are dry or rough from deposit.

Sounds produced by Succussion:—

A Splashing Sound, when the cavity of the pleuræ, or a large tuberculous cavity, contains fluid mixed with air.

Sounds produced by the Contraction of the Muscles:—

Vibratory Sounds of varying intensity.

TABULAR VIEW OF THE THORACIC REGIONS IN RELATION TO THE SIGNS OF PERCUSSION AND AUSCULTATION.

REGIONS.		SITUATION.	NATURAL SOUND ON PERCUSSION.	INTERIOR CORRESPONDING PARTS.	SIGNS MOST COMMONLY PRODUCED THERE BY DISEASE.
1. Clavicular.	2.	Clavicles.	Very clear towards the sternum; clear in the middle; dull close to the humerus.	Apices of the lungs.	Dulness on percussion in phthisis; generally most on one side.
2. Infraclavicular.	2.	Between the clavicles and the fourth ribs.	Very clear.	Superior lobes of the lungs, large bronchi near the sternum.	Irregular dulness on percussion, diffuse bronchophony, impaired respiration, and afterwards cavernous rhonchus, and pectoriloquy, in phthisis. Various rhonchi in catarrhs.
3. Mammary.	2.	Between the fourth and eighth ribs.	Very clear; particularly by mediate percussion.	Middle lobes of the lungs; large bronchi in the upper part, near the sternum; the heart, generally	Rhonchi in catarrh; more rarely phthisical symptoms. On the left side, dulness on percussion in hydropericardium and enlargement of the heart; increased impulse in hypertrophy and increased

[TABLE—*Continued.*

TABULAR VIEW OF THE THORACIC REGIONS—*Continued.*

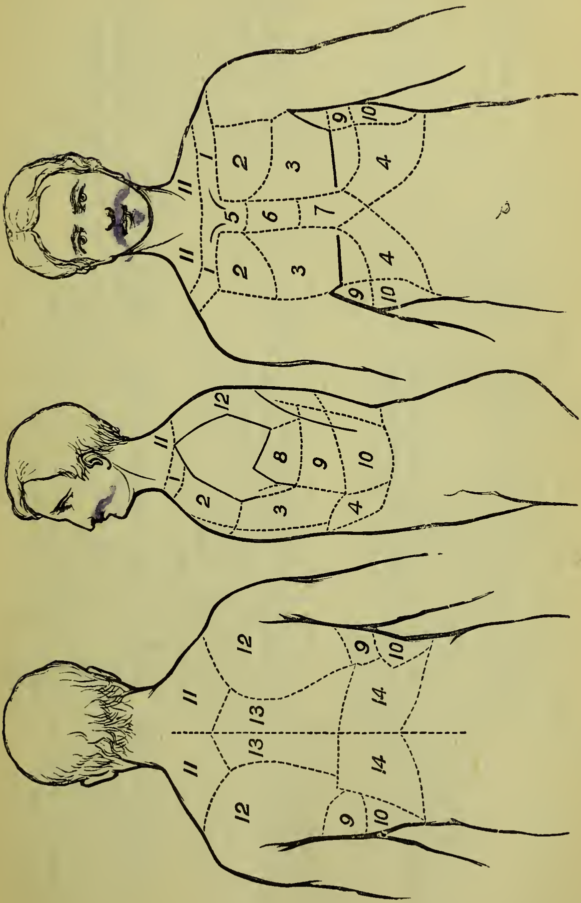
REGIONS.		SITUATION.	NATURAL SOUND ON PERCUSSION.	INTERIOR CORRESPONDING PARTS.	SIGNS MOST COMMONLY PRODUCED THERE BY DISEASE.
				covered by the lungs, in the lower part of the left region.	sound of pulsation in dilatation of the heart; constant rasp; a bellows sound in valvular disease.
4. Infra-mammary.	2.	Between the eighth ribs and the margin of the cartilages of the false ribs.	Dull on the right side; on the left irregularly dull or unnaturally resonant.	The liver on the right, and the stomach on the left, slightly covered by lungs.	Crepitant rhonchus in incipient pneumonia. Extinction of respiration in advancing pleurisy. Dry crepitation in interlobular emphysema.
5. Superior sternal.	1.	Upper parts of the sternum.	Very clear.	Large bronchi.	Bronchial rhonchi in catarrh. Only half the sternum dull on percussion in hepatisation, the whole dull in extensive liquid effusion of one side.
6. Middle sternal.	1.	Middle part of the sternum.	Very clear.	Margins of the middle lobes of the lungs.	
7. Inferior sternal.	1.	Lower part of the sternum and ensiform cartilage.	In the upper part clear, rather less so in fat persons. Below sometimes more dull, sometimes tympanitic.	Above, margins of the lungs; below, the heart, liver, and sometimes the stomach.	Signs of disease of the right side of the heart; dulness on percussion in effusion, or fat in the pericardium, enlarged heart, etc.

8. Axillary.	2.	In the axilla above the fourth ribs.	Very clear.	Upper part of the lateral lobes of the lungs. Large bronchi.	Dulness on percussion, cavernous rhonchus, pectoriloquy, etc., in phthisis. Catarrhal rhonchi.
9. Lateral.	2.	Between the fourth and eighth ribs at the sides.	Very clear, unnaturally so in emphysema of the lung.	Middle of the lateral lobes of the lungs.	Dulness on percussion in advanced pleurisy, and on the right side from enlarged liver; Ægophony in advancing pleurisy; crepitant rhonchus and bronchophony in advancing pneumonia.
10. Inferior lateral.	2.	Below the eighth ribs at the side.	The same as the infra-mammary.	Margin of the lateral lobes of the lungs; the liver on right, and the stomach and spleen on the left side.	Crepitant rhonchus in incipient pneumonia. Extinction of respiration in pleurisy.
11. Acromial.	2.	Between the clavicles and upper margin of the scapulæ.	Dull by direct percussion. A tolerably clear sound may be elicited by mediate percussion, particularly near the clavicle.	Superior lobes of the lungs and large bronchi.	Dulness on percussion in extensive tubercular accumulation, cavernous rhonchus and respiration and pectoriloquy in phthisis. Catarrhal rhonchi.

[TABLE—*Continued.*

TABULAR VIEW OF THE THORACIC REGIONS—*Continued.*

REGIONS.		SITUATION.	NATURAL SOUND ON PERCUSSION.	INTERIOR CORRESPONDING PARTS.	SIGNS MOST COMMONLY PRODUCED THERE BY DISEASE.
12. Scapular.	2.	The scapulæ and the muscular ridge below them.	The pectoral resonance can be elicited from this region only by mediate percussion.	Middle posterior lobes of the lungs.	Catarrhal signs. Ægophony in pleurisy. Bronchophony in pneumonia.
13. Interscapular.	2.	Between the inner margin of the scapulæ.	Pretty clear by mediate percussion, or when the arms are crossed and the head bowed forwards.	The roots and inner parts of the posterior lobes of the lungs.	Catarrhal signs. In the upper part, sound of respiration never destroyed in effusions into the pleura. In the lower portion, sometimes ægophony in pleurisy, crepitation and bronchophony in advancing pneumonia. Signs of diseased bronchial glands.
14. Inferior dorsal.	2.	Below the inferior angles of the scapulæ and border of the serrati to the level of the 12th dorsal vertebra.	Clear on the upper portion, by striking on the angles of the ribs, or by mediate percussion. Below, dull on the right, and tympanitic on the left side.	Base of the lungs. The liver encroaches on the right, and the stomach on the left side.	Crepitant rhonchus and bronchophony, in incipient pneumonia and œdema; ægophony, in pleurisy; and dulness on percussion in both.



MORBID THORACIC SOUNDS HEARD DURING RESPIRATION.

	CHARACTER AND SITE OF SOUND, CAUSE, ETC.	RELATION TO INSPIRATION AND EXPIRATION.	PATHOLOGICAL SIGNIFICATION.
1. BUBBLING RHONCHI.	The passage of air through mucus, blood, pus, or serum, in the bronchial tubes, or in cavities communicating with them.		
(*a*) Large bubbling rhonchi. *Syn.*—Mucous râle.	Bubbles of large but of unequal size, bursting irregularly in the larger bronchial tubes. Heard more or less all over the chest, but more particularly over the central and lower part of the lungs. Modified by coughing and expectoration.	Heard with inspiration and expiration.	Bronchitis, when there is free secretion. May be heard also in cases of hæmoptysis, when there is blood in the larger bronchi.
(*b*) Small bubbling rhonchus. *Syn.*—Sub-mucous râle, muco-crepitating rattle, rhonchus subcrepitans.	Bubbles small, unequal in size, irregular, and in small bronchi. Heard over lower parts of lung in capillary bronchitis. Apex in early stages of phthisis, and more extended during the stage of resolution in pneumonia.	Heard with inspiration and expiration, but most loudly during the former.	Capillary bronchitis, phthisis, and in pneumonia during resolution.

(*c*) Hollow bubbling rhonchus. *Syn.*—Cavernous râle, gurgling rhonchus.	Bubbles few and large, bursting with a hollow gurgling sound in an enclosed space.	Heard with both inspiration and expiration.	Excavation of the lung in phthisis. Dilatation of a bronchus, or broncho-pleural fistula.
2. CRACKLING.			
(*a*) Moist crackling rhonchus. *Syn.*—Humid crepitation, clicking, humid crackling rhonchus.	A series of smart moist clicking sounds of nearly equal intensity and duration. Heard in all parts of the chest. Cause obscure.	Occurring during the whole of inspiration and the first half of expiration.	Disintegration and breaking down of tubercular deposit.
(*b*) Dry crackling rhonchus. *Syn.*—Dry crepitation, crackling.	A series of short dry crackling sounds. Heard just below or above the clavicles. Cause uncertain.	Present *only* during inspiration.	Commencing softening of phthisical consolidations.
3. CREPITATION.			
(*a*) Primary crepitating rhonchus. *Syn.*—Crepitation, crepitant râle, intravesicular râle.	A continuation of fine sharp dry sounds of equal duration and intensity. Intensified by deep breathing and coughing. Dr Williams says, "It may be tolerably represented by rubbing transversely between the fingers and thumb a lock of one's own hair close to the ear." Due probably to forcible distention of inflamed air vesicles.	Occurs only with inspiration, and often then only heard at its close.	Early stage of pneumonia or pneumonic inflammation round tubercle.

[TABLE—*Continued.*

MORBID THORACIC SOUNDS HEARD DURING RESPIRATION—*Continued.*

	CHARACTER AND SITE OF SOUND, CAUSE, ETC.	RELATION TO INSPIRATION AND EXPIRATION.	PATHOLOGICAL SIGNIFICATION.
(*b*) Secondary crepitating rhonchus. *Syn.*—Rhonchus crepitans redux, redux crepitation.	Somewhat like the above, but moist, fewer, and unequal in intensity and duration. May be due to air passing through fluid in minute bronchi.	Audible both during inspiration and expiration, but chiefly during the former.	Marking the stage of resolution in pneumonia.
4. VIBRATING.			
(*a*) Sonorous rhonchus. *Syn.*—Sonorous râle.	A musical sound of low pitch, cooing in character, sometimes snoring, equable, continuous, and varying in intensity and duration. Fremitus of the thoracic walls attends it. Probably in larger bronchi.	Occurs with both inspiration and expiration, but chiefly the latter, to which it may be entirely limited.	Bronchitis.
(*b*) Sibilant rhonchus. *Syn.*—Sibilant râle.	A hissing sound of high pitch. Whistling or piping. Air passing through viscid mucus or narrowed bronchi.	Co-existent with inspiration and expiration; more marked with the former, but may be limited to either.	Emphysema, bronchitis, and spasmodic asthma.

The Physical Signs which distinguish Bronchitis, Pneumonia, and Pleurisy from one another are the following:—

	BRONCHITIS.	PNEUMONIA.	PLEURISY.
Percussion...	Resonance of health.	Slight or moderate dulness, increasing with the severity of the disease.	Dulness, when effusion has taken place.
Auscultation.	The dry and moist sounds absent at first outset of the disease, become more marked in a later stage.	Crepitant râle in the first stage. When solidification occurs, *bronchial respiration*, *bronchophony* and *whispering bronchophony* are the signs.	A rubbing "to and fro" sound heard during respiration.
Sputa.........	At first, glairy frothy mucus, becoming thicker and of a yellowish or greenish colour.	At first, scanty, clear, and viscid; then semi-transparent, adhesive with a reddish tint, like that of iron rust; the colour due to blood corpuscles.	None, unless complicated with bronchitis or pneumonia.
Pain..........	Dull, when present, with a feeling of soreness and rawness.	Sometimes absent, at others acute.	Acute pain in the side. This is not diagnostic, as it may occur in intercostal neuralgia and pleurodynia.
Part affected.	Bronchi and air tubes.	Parenchyma of the lung.	The pleuræ covering the lung.

BRONCHITIS.

Lat. *Bronchitis.* Fr. *Bronchitè.* Ger. *Bronchitis.*

DEFINITION.—An inflammatory affection of the mucous membrane of the bronchial tubes, the diseased action extending into the pulmonary vesicles.

Bronchitis has been divided into acute, chronic, and plastic or croupous bronchitis. The following are among the predisposing causes of this disease:—

(*a*) Early childhood, especially during the period of dentition.

(*b*) Old age.

(*c*) Improper food, and faulty hygienic conditions.

(*d*) Previous bronchial attacks.

The exciting causes may thus be tabulated:—

(*a*) The circulation in the bronchial vein may be impeded, giving rise to hyperæmia of the lungs.

(*b*) The same condition may occur if any impediment be placed on the circulation of the great vessels of the aorta.

(*c*) Irritants acting directly on the mucous membrane of the air tubes. Coal heavers, stone cutters, millers, and others engaged in like trades frequently suffer from this disease. The terms applied to bronchitis (phthisis?) occurring among coal heavers is *anthracosis* (ανθραξ, a coal); in workers with oxide of iron, *siderosis* (σιδηρος, iron); with sandstone, clay, etc., *chalicosis* (χάλιξ, gravel).

(*d*) Sudden chilling of the external surface of the body.

(*e*) It may form one of the complications of gout, rheumatism, typhoid, or any of the eruptive fevers.

(*f*) Certain not well-ascertained atmospheric changes may give rise to epidemic forms of this disease.

Symptoms.—The symptoms which usher in an attack of acute bronchitis are those which commonly attend a "cold"—chilliness, flushes of heat, quick pulse, more or less thirst, furred tongue, urine loaded with phosphates, etc. The respiration, as the disease becomes more pronounced, is hurried; there is a sensation of tightness, accompanied with the feeling of soreness about the chest. Pain, when present, generally follows a fit of coughing, and is of a dull aching character, felt beneath the sternum. The pulse is quickened; regular, but feeble. The temperature varies from 98° to 99·5° F.; if it rise higher, some complication may be suspected. The cough is at first dry, but is soon accompanied with the expectoration of a varying quantity of clear, frothy, viscid mucus sometimes streaked with blood, and which is at first slightly saline to the taste. When the expectoration possesses these characters the inflammation is still active. Co-incident with a diminution in the inflammatory action, the matter expectorated becomes thick, and of a yellowish or greenish colour. Sometimes the expectoration is in the form of pellets, or oyster-looking masses. A return to the frothy sputa points to the recurrence, increase, or extension of the inflammatory action. As long as the inflammatory process is confined to the larger bronchial tubes, the disease though severe is not necessarily fatal; but when the smaller tubes are affected—"capillary bronchitis,"—the danger to life is greatly increased. The symptoms then become aggravated, the dyspnœa is constant, and alarming in proportion as the smaller tubes become implicated. The face is pale, the eyes suffused with tears, the nostrils dilate during inspiration, and the countenance expresses great anxiety. Sometimes the face is bloated and of a bluish colour, due to the impaired circulation through the lung. The patient ultimately dies of apnœa, cold clammy sweats and sometimes delirium ushering in the fatal termination.

The physical signs of bronchitis are important. Percussion gives no direct result, for the resonance of health is unimpaired. This is only to a certain extent true, for if the lung be emphysematous, there will be increased resonance; if, on the other hand, a portion of the lung be deprived of air from a plug of mucus in a bronchial tube, there will be dulness due to collapse of the lung. In the majority of cases, palpation detects a vibratory tremor. The results of auscultation in the early stage are often negative. After a time, however, the respiratory sounds become modified, or entirely masked by the different râles. The sounds now heard are distinguished into the *dry* and the *moist.* Obstruction of the bronchial tubes due to the tumefaction of their lining membrane gives rise to the first; whereas the latter are caused by the presence of mucus, through which the air passes in its entrance into, and exit from, the lung. The *dry* sounds when situated in the large tubes are called *rhonchus;* when in the smaller, *sibilus.* The latter, pointing to implication of the smaller air tubes, betoken the presence of danger to the lung tissue and probable consolidation. The term *crepitus,* which may be either *large* or *small,* having reference to the size of the tubes in which it occurs, is applied to the moist sounds. These sounds are heard when exudation has taken place into the tubes, and are caused by the air bubbling through the exuded mucus. It should be borne in mind that the respiratory murmur over a portion of the chest may be temporarily suppressed, owing to the obstruction of a bronchial tube by mucus. Collapse of a portion of the lung may also follow from this cause, and as a result, other parts, chiefly the anterior and upper lobes, may become emphysematous, due to the extra strain on the portions of the lung left free to act. Should, during an attack of bronchitis, dulness over a portion of the chest accompanied with laborious long-drawn exhausting respiration occur, collapse of the lung has taken place. Death may follow so soon after a bronchus has become plugged, that collapse of the lung has not had time to occur; in that case, all respiratory murmur ceases, although the percussion sound remains unaffected.

Of the pathology and mechanism of pulmonary collapse it is necessary here to say a few words. Pulmonary collapse is closely allied to that condition of the lung (*atelectasis*) found in the imperfectly respired lung of new-born infants. The condition of the lung in infants, however, more closely resembles the "carnification" of the lung caused by compression by pleural effusion than the condition of collapse, for the fœtal state is persistent, the lung never having been inflated. To account for the condition of collapse, Dr Gairdner suggested that a portion of mucus is forced by every inspiration from a large into a smaller bronchial tube; expiration again moves it forward, allowing all the air to escape from the air cells beyond the obstruction; another inspiration forces it further down, and this process is continued till all the air has been expelled from the air cells beyond the plug, and collapse of the portion of lung thus cut off is the result. The action of the plug is thus that of a "ball valve" allowing egress but not ingress of air. Now, allowing that this is the primary cause of collapse, there must come a time when, owing to the elastic force of the lung tissue, the confined air is incapable of moving the plug. How then is the air removed and the lung collapsed? May not the air, as suggested by Lichtheim of Berne, be absorbed by the blood which circulates in the pulmonary alveoli. And that this is the probable explanation is shown by the above mentioned observer who has demonstrated that the elasticity of the lung is not destroyed till all the air from the lung has been removed.

Acute bronchitis, when not complicated with other disease, is not usually fatal, except when it occurs in the very young, the old, and the delicate, and then it is a most fatal disease, ranking next to phthisis and before pneumonia in its fatality. The rapidly fatal cases in young persons are probably due, as suggested by Dr Wilks, to acute tuberculosis, and not to bronchitis, which he considers to be seldom fatal unless complicated with other diseases, or when an acute attack follows upon a previous or semi-chronic attack. In these subjects the danger is due to plugging up of the bronchial tubes by inflammatory products. Death then occurs from apnœa. In children, in many cases, an attack of convulsions closes the scene, a termination most common in all acute diseases in children. You will often have babies brought to you during the period of teething, and be told that the child always cuts its teeth with bronchitis or "a cold in its chest." I do not believe that these cases are cases of true bronchitis; and for this reason, that you will in most cases find that, as soon as the tooth is through, the bronchitis goes, to return at the advent of the next tooth. The bronchitic symptoms are probably due to irritation of the vagus nerve.

Post-mortem Signs.—The lungs are found distended, and do not collapse when the sternum is removed; the trachea and bronchi are also more or less blocked up with frothy adhesive mucus. The mucous membrane of the tubes is thickened and red.

Treatment.—The treatment of uncomplicated acute bronchitis is simple enough. At the very onset of the disease, that is, during the catarrhal stage, a diaphoretic, combined with a full dose of opium, may arrest, or at least modify, the bronchial attack. It is usual to give ten grains of the compound ipecacuanha powder, in some hot gruel, just before going to bed; to have the feet placed in hot water, to be followed in the morning by the administration of a saline aperient. Should this not succeed in arresting the disease, the patient, in severe cases, should be kept in a moist atmosphere at a uniform temperature. The inhalation of steam often affords intense relief, and a patient at one moment scarcely able to breathe may, by the mere inhalation of the steam of boiling water, be restored to comparative comfort. The application of hot poultices, or a stimulating liniment to the chest, often affords great relief. Cupping between the shoulders, or the application of a blister to the front of the chest, is very useful in some cases. Sir Thomas Watson strongly recommends mercury and antimony in the acute stage of bronchitis. Tartar emetic is useful in the early stages, and should be more frequently used. Ipecacuanha is also a useful remedy, as it appears to have the power, when absorbed, of liquefying the mucous secretion, and thus assisting its ready expectoration.

Expectorants, when the expectoration becomes loosened, may be given with advantage; those most usually employed are squill, ipecacuanha, tartar emetic, the carbonate of ammonia, and senega. When necessary, stimulants must be freely used.

CHRONIC BRONCHITIS.

Lat. *Bronchitis longa.* Fr. *Bronchite Chronique.*
Ger. *Bronchitis chronische.*

Definition.—Chronic inflammation of the bronchial tubes.

This disease seldom occurs except in those who have passed middle life, and is then sometimes spoken of as "winter cough." It may occur, however, in the young as the sequel of the acute form of the disorder. It may accompany gout, heart disease, or chronic Bright's disease.

Symptoms.—The primary symptoms are those of acute bronchitis, but less severe. The expectoration is sometimes profuse, often coughed up in pellet-shaped masses. The cough is troublesome, and may or may not be accompanied with profuse expectoration. In some cases the patient makes ineffectual efforts to "bring up the phlegm," *catarrhe sec*, or "dry bronchitis;" in others, the expectoration is excessive, constant, and annoying, *bronchorrhœa serosa.* He will often express his wonder "where it all comes from." Percussion is normal, or there is slight emphysema with increased resonance. *Emphysema* attended with great dyspnœa is often a result of repeated attacks of chronic bronchitis. When dilatation of the bronchi, or bronchiectasis, is present, the following conditions and symptoms are present:—The bronchus is dilated, *cylindrical* or *sacculated*, and in the dilated portion, mucus collects; this may become purulent. In cylindrical bronchiectasis the dilated tubes may run through the lungs, with slight, if any, diminution in their calibre, till they reach the pleural surface, not unlike the distended fingers of a glove. In the sacculated variety, "when a section is made of the lung, the cut surface appears to be covered with an immense number of shallow, smooth-walled depressions, exactly like so many minute saucers." These conditions are doubtless brought about by the mechanical action of respiratory pressure, analogous to that producing emphysema. A characteristic symptom of this condition is that after a severe fit of coughing the patient expectorates a large quantity of semi-purulent mucus, followed by relief. These attacks frequently occur night and morning. Beware of mistaking a dilated bronchus for a vomica; the mistake has been made, and may be made again. Examine into and look for other signs of phthisis, history, emaciation, etc.

The **Post-mortem** appearances of this form of bronchitis are those of inflammation generally. The air tubes are choked up with tenacious mucus, sometimes with a muco-purulent fluid. In all cases examine carefully the smaller bronchial tubes by slitting them up. You will then find enough to account for death where at first sight nothing appeared. Here and there a portion of the lung may have entirely collapsed. Dilatation of the bronchi—bronchiectasis—together with emphysema, are very commonly found after death.

This disease must not be mistaken for pulmonary œdema, emphysema, or chronic pneumonia.

Treatment.—Before adopting any particular mode of treatment, the general condition of the patient should be considered. If weak and debiliated, a generous, easily digested diet should be enjoined, with tonics. Ammonia, squill, senega, and other expectorants may be administered, F. ~~16~~. 24 The balsam of copaiba, the muriate of ammonia, and the chlorate of potash, have been largely recommended. The dyspnœa may be best relieved by the inhalation of steam, or steam impregnated with benzoin, compound tincture of camphor, etc. etc. In the gouty, or those who have suffered from syphilis, colchicum or the iodide of potassium should be given with the other medicines used. A residence in South Devon, or other place having a warm climate, is often better than drugs in the cure or relief of chronic bronchitis.

PLASTIC, OR CROUPOUS BRONCHITIS.

Lat. *Plasmata bronchiorum.* Fr. *Bronchite pseudo-membraneuse.* Ger. *Abdrücke der Bronchiadröhren.*

Definition.—An exudation of coagulable lymph on the mucous lining of the inflamed bronchi.

This is a form of bronchitis of extreme rarity. It may exist *per se*, or it may be simply an extension of a croupous or diphtheritic state of the trachea or larynx.

The symptoms are much the same as those of acute bronchitis.

The only diagnostic sign of this disease is, of course, the expectoration of the false membrane which is often expectorated as casts of the bronchial tubes, varying considerably in length.

The treatment is the same as that adopted in the other forms of bronchitis, but does not appear to be very successful. The iodide of potassium has been given with apparently good results.

ASTHMA.

Lat. *Asthma.* Fr. *Asthme.* Ger. *Asthma.*

Definition.—A specific disease characterised by spasmodic contraction of the bronchial tubes and air vesicles, with difficulty of breathing resulting therefrom.

This disease, probably having its origin in certain morbid states of the nervous system, consists in the obstruction of the bronchi, especially the smaller tubes, by tonic spasm of the organic muscular fibres that enter into their construction. The predisposing causes of this affection are very obscure, but there is every reason to believe that asthma is often hereditary. It may occur in children as well as in adults, in males more frequently than in females. The exciting causes in most cases appear to depend on atmospheric changes. Certain localities also, it would seem, possess a peculiar obnoxious power over those predisposed to asthma.

Symptoms.—The attack comes on suddenly, perhaps in the middle of the night, waking the patient from his sound sleep. Laborious breathing, attended with great dyspnœa, characterises a paroxysm. Every effort is made to breathe, the respiratory muscles are brought powerfully into action, the nostrils are dilated, the head thrown backwards, and the whole expression of the face is one of extreme terror. The act of inspiration is attended with a spasmodic effort, and expiration is prolonged and accompanied with a wheezing sound. The proper respiratory sounds are absent, and in their place we get sibilant râles, rhonchi, and a peculiar wheezing hissing sound. An abnormal resonance is detected on percussion. The face becomes pale, often assuming a bluish tint. The voice is almost lost for want of breath. The pulse is small and feeble, and the surface of the body is often bathed in a cold clammy sweat. At last a fit of coughing takes place, accompanied with a free discharge of mucous, and the patient is relieved.

The **Post-mortem** appearances, in cases where the disease proves fatal, are those of bronchitis and emphysema, with which it is always more or less accompanied. Leyden, in 1871, discovered in the sputa pointed octohedral crystals, supposed by some to be the cause of asthma by irritating the vagi in the bronchial mucous membranes, but they are also found in sputa of persons not suffering from asthma.

The **Diagnosis** is not difficult when the symptoms as just described are taken into consideration. As far as immediate danger is concerned, the prognosis is far more favourable than the severity of the symptoms would lead one to expect.

Treatment. — The indications for treatment are, first, to diminish as much as possible the suffering, to mitigate and shorten the paroxysms, and then to take measures to prevent their recurrence. In our attempts to stop the paroxysm, the past experience of the patient will be of great assistance. The inhalation of ether or chloroform, the smoke from brown paper, or a pipe or two of stramonium are often of great service. In cases uncomplicated with bronchitis, a full opiate may be given; in others, nauseant remedies are found of most use. If the attack has been induced by an overloaded stomach, an emetic or brisk purgative is indicated. In all cases remove, if possible, any concurrent affection, dyspepsia, gout, etc. Treat the diathesis, and you will often succeed, if not in curing the patient, at least in giving him great relief and enjoyment in life. The preventive measures consist in removal from the obnoxious locality, and from those influences which experience has shown to be peculiarly prejudicial.

CONGESTION AND ŒDEMA OF THE LUNG.

Congestion occurs as one of the phenomena of inflammation, but simple congestion of the lung is not of unfrequent occurrence, and often complicates other morbid conditions. Congestion may be the result of mechanical causes, and it may also be either *active* or *passive.*

Active congestion may be produced by irritating substances applied to the part, or by an obstruction suddenly acting on the vessels.

Passive congestion differs from the active form more in degree than in kind. Among the most frequent causes of passive congestion is disease of the valves of the heart or general weakness of the organ. In fevers, and in diseases accompanied with extreme prostration and debility, passive congestion is a frequent and severe complication. As a result of passive congestion of the lungs we may also have œdema, but this condition may also be the local expression of a general dropsy.

For an account of the pathological condition of the congested lung the reader is referred to the description of "Pneumonia." The symptoms and physical signs are also those of the early stage of that disease. In practice, congestion of the lung is not unfrequently met with, so slow in its production, that little information as to its cause can be obtained from the history of the case. Some slight exposure to cold may be mentioned, but the slight cough and scanty expectoration give no index to the condition of the lung found on percussion and auscultation.

I have a case now under treatment in which the whole of the left lung was congested, and which had been previously treated, before I saw the patient, as a case of debility. The lung is gradually improving; rest in bed, blistering and painting the chest with the iodine liniment, and the administration of the ammonio-citrate of iron and iodide of potassium, have been the means adopted in the treatment of the case.

In the general treatment of simple congestion of the lung, the cause, heart disease, etc., must be sought and removed.

PLEURISY.

Lat. *Pleuritis.* Fr. *Pleurésie.* Ger. *Pleuritis.*
Syn. *Reppenfellentzündung.*

INFLAMMATION OF THE PLEURÆ.

DEFINITION.—Inflammation of the serous membrane or pleuræ which surround the lungs.

Causes.—Pleurisy may be produced by injuries to the walls of the chest, such as fracture of the ribs, or by penetrating wounds. It may also be due to the effects of *cold*, or it may occur as a concomitant in Bright's disease of the kidney, acute rheumatism, and as a sequel to the eruptive fevers. It is rare in infancy and old age, being more frequently a disease of youth and middle life. The chronic or sub-acute form is less often met with than the acute.

An attack of acute pleurisy may be divided into three stages:—

1. The stage of inflammation.
2. The stage of effusion.
3. The stage occupied in the removal of the effusion.

For a day or two previous to the first stage, some amount of pain or tenderness may be experienced in the region of the chest. The patient may also complain of "feeling out of sorts" and somewhat below par; in fact, just in that condition best adapted for the reception of morbid influences. Under these conditions, if a sudden chill be experienced by the patient, bronchitis, pleurisy, etc., may occur; if exposed to the poison of typhoid fever, that disease may follow; and so on.

The *Stage of Inflammation* sometimes sets in with a well-marked chill, followed by rigors; at others, only a sensation of chilliness is experienced. Intense pain of a cutting or a stabbing nature, augmented by the act of inspiration, is felt early in the inflammatory stage. The pain is sometimes described as a "stitch in the side," and is generally felt close under the mammary gland. Coughing, sneezing, or the slightest movement of the body, tends to increase the pain. Acute pleurisy is always attended with moderate pyrexia, and the usual signs and symptoms of fever are present. The cough is dry and short, and the act of coughing is suppressed as much as possible. Expectoration is slight, consisting chiefly of simple mucus. The respirations are increased in frequency, and are much shortened by the patient. If the patient be requested to take a deep breath, it will be found that he begins cautiously to take a deep inspiration till he reaches a certain point, when suddenly he stops with a jerk. If the inflammation attack the diaphragmatic pleuræ, pain is felt beneath the ensiform cartilage, the respirations are restrained, and troublesome hiccough annoys the patient. Vomiting may also occur, and the patient complains of acute pain at the base of the thorax. During this stage the pleuræ are undergoing well-marked changes. There is hyperæmia, a rapid increase in the blood and epithelial cells, and a pouring out of liquor sanguinis. "The endothelial cells enlarge and become more granular, their nuclei multiply, and several new elements are formed within a single cell (endogenous multiplication), from which they subsequently escape." A layer of lymph now forms a thin film over the surface of the pleuræ, which may glue the two surfaces together. At first this film may be easily pulled off, but it soon becomes fibrous, causing the lung to adhere to the chest. Soon after these changes begin, the pleuræ loses its smooth and glistening appearance, becoming opaque and rough. If the inflammatory action be not early arrested, fluid is poured out which may become purulent, and an *empyema* result. This may, however, be due to constitutional causes, and is far more frequently found among the strumous and weakly than among the robust.

The physical signs of the stage prior to that of effusion are the well-known *friction sounds*, caused by the rubbing together of the inflamed surfaces of the pleuræ. These sounds soon cease, their cessation being due to one of three causes: the inflammation may terminate in resolution; the inflamed pleuræ may become glued together by a process known as *adhesive inflammation;* or, an effusion of serum may take place into the pleural cavity, and a condition known as hydrothorax may ensue, or we may have an empyema.

The *Stage of Effusion* is generally marked by a diminution of the acute symptoms: the pain is lessened; the act of coughing is not attended with the acute pain of the first stage; the respirations are still more or less quickened, the quickening depending on the amount of effusion and compression of the lung. The affected side may bulge out from the effusion.

The patient usually lies on the affected side or on his back, but if the quantity of the effusion be large, dyspnœa may occur, and the patient may find it impossible to maintain a recumbent posture. The dyspnœa is regulated by the amount of fluid present, and is thus a more or less certain index of the condition of the patient, and the amount of lung compression.

When suppuration takes place within the pleural cavity, and pus is formed, a condition known as *empyema* ensues. Empyema may be *true* or *false*—*true*, when due to inflammation of the pleuræ; *false*, when caused by the rupture of an abscess in the lung, and escape of its contents into the pleural sacs.

The physical signs which mark the stage of effusion are—diminution of the respiratory sounds, the vesicular murmur becomes almost inaudible, bronchial breathing is heard, and *bronchophony*, or bronchial voice, is also present. Bronchophony is often attended with a peculiar bleating sound, known as *ægophony*. Dr Stone has shown that the cause of ægophony is due to the fact "that the fundamental tone is interrupted in its passage through a layer of pleural exudation, while the overtones are allowed to pass and, being heard by themselves, give the peculiar character to the sound." It has also been shown that the French *a* yielded hardly any ægophony, but that ægophony became more marked with *e*, *i*, *u* placed in the order of their producing power of the sound. Puerile breathing, due to the increased activity of the lung on the healthy side, is heard.

When the effusion is very great, no respiratory murmur is heard: and the diminution, or entire absence of the vocal thrill, or "tactile vibration," distinguishes pleurisy even with *moderate* effusion from pneumonia, in which latter disease the *vocal thrill is increased*. Due to the presence of fluid, the heart may be considerably displaced. The amount of dulness on percussion will depend on the quantity of the pleuritic effusion; and when this is moderate in quantity, the tympanitic sound is observed above the level of the fluid in the anterior and lateral parts of the thorax. Not in every case, however, is the sound tympanitic above the fluid, due probably to the unequal tension of the lung-tissue in different persons. It must be borne in mind also, that, although fluid gravitates to the lowest part of the thorax, it is not always found there, for old attacks of pleurisy may have glued the lower portions of the pleuræ together, thus raising the base of the fluid collection. It is not improbable that in some cases the fluid may even become sacculated. As a result, too, of fluid pressure, the lung becomes compressed, and this compression may lead to an entire *carnification* of the lung, the lung becoming *bloodless* as well as *airless*—a condition differing from *collapse*, in which the lung is *only airless*. The fluid need not be large in quantity, a few ounces will suffice to compress and cause a total collapse of the pulmonary tissue which ought to occupy that space. The patient now generally prefers to lie on the affected side; in the first stage of the disease he may recline on the healthy side.

The *Stage of Absorption* is marked by the disappearance of the fever, and by a marked freedom from pain and cough. The respirations gradually approach the normal standard as the effusion becomes absorbed. The physical signs of health become more and more apparent.

Pleurisy is an unilateral disease, most frequently affecting the pleura of one side only, and then usually the left.

Diagnosis.—The pain is not, when taken alone, a diagnostic symptom, for it is present in intercostal neuralgia and pleurodynia, and in shingles; pleurisy may, however, be overlooked, when the pain is but slight. In pleurodynia the pain is, as a rule, constant and not increased during a prolonged inspiration, and there is an absence of fever and friction sounds. The presence of the "friction sound" is important, but the rubbing of tubercles projecting above the surface of the pulmonary pleura has been mistaken as due to pleurisy. The crepitant râle of pneumonia will at once distinguish pleurisy from pneumonia. And in pneumonia the sense of resistance to the finger on percussing the chest is not so great, nor is the tonelessness so marked as in fluid effusion. During the stage of effusion there will be marked dulness on percussion, modified by the position in which the patient is placed, the fluid gravitating to the lowest point. But this latter sign, as we have just shown, may be modified by varying conditions within the thorax. Moreover, it is not easy, at least not so easy as many young students appear to think, to be able to detect the presence of small quantities of fluid; and experienced observers have declared that eight or ten ounces of fluid, a quantity quite sufficient to give rise to distressing symptoms, can "scarcely be detected with certainty." Do not mistake *hydatid* in the liver for right-sided pleurisy or large chronic *pericardial effusion.* This error is not uncommon. Malignant disease in the thorax has been time and again mistaken for chronic pleuritic effusion. Empyema may be suspected if the pleurisy be of long duration and there be repeated rigors.

Prognosis.—In simple acute pleurisy the prognosis is, as a rule, favourable; yet cases of sudden death are by no means rare if the effusion be large, and this termination appears to be more frequent in pleurisy of the left side, which, by the way, is its most common situation. It has been suggested by Bartels of Kiel, that when the heart is pushed over to the right side the inferior vena cava is bent almost to a *right* angle close to the diaphragm, and further covered by the wall of the auricle.

Treatment.—In the first stage, if the patient be robust and plethoric, with a strong non-compressible pulse, and suffering from great pain and oppression, general bleeding will afford great relief. Dry or wet cupping may be resorted to, or the application of leeches may prove useful. Hot linseed-meal poultices, or the application of a blister to the chest, should be used, if general or local bleeding be deemed unadvisable. In many cases a blister will be found to act like magic. All the mustard plasters you may apply have not the effect of a single blister. I have often tried mustard, and when obliged to have recourse to a blister regretted that I had not applied one in the first instance. Even if the patient be not very strong, a blister will often, I am sure, cut short the disease. Opium, in doses large enough to relieve pain, is not contra-indicated. The compound powder of ipecacuanha at bed-time is most useful. The hypodermic injection of morphia may be used if the pain be very severe. Saline purgatives, combined with medicines which have a sedative effect on the circulation, should be given at the very outset of the disease. The tincture of aconite may be given in three-minim doses, or a mixture containing the acetate of ammonia, vinum antim: tart: and sulphate of magnesia may be given.

To cause absorption of the effusion, drastic purgatives may be used with care; the application of blisters, or painting the chest with the tincture of iodine, or the inunction of mercurial ointment, may be tried; and of the external use of the latter the late Dr Fagge speaks with great confidence, especially when combined with the internal administration of the iodide of potassium. The iodide of lead ointment may be rubbed into the chest. Diuretics, the acetate of potash, combined with squill and digitalis, are sometimes given, but their value is doubtful. The strength of the patient should be supported by a nourishing diet. Recourse should at once be had to the operation of paracentesis thoracis, as soon as the presence of fluid in large quantity is distinctly made out, the presence even of high temperature does not contra-indicate the performance of the operation. When the fluid is small in quantity, the operation may be delayed a few days; but the patient will require careful watching, for the reasons given under the prognosis of the disease. All the fluid need not be drawn off at once, and the slower the better.

CHRONIC PLEURISY.

Lat. *Pleuritis longa.* Fr. *Pleurésie chronique.*
Ger. *Chronische Pleuritis.*

DEFINITION.—Chronic inflammation of the pleuræ, either as a primary or secondary affection, following an acute attack.

This disease may occur as a sub-acute affection from the first, or it may follow an acute attack of pleurisy. The symptoms are less marked than in the acute form; and like causes are instrumental in the production of both the acute and chronic forms of pleurisy. The treatment is the same as in the acute form; but the patient will require the administration of tonics, and the adoption of supporting measures.

THE SEVERAL KINDS OF EFFUSION WHICH MAY BE FOUND IN THE PLEURAL SAC.

(*a*) *Serous.*—An effusion of serum of a yellowish-green or straw colour, containing particles of coagulated fibrine, and occurring during the course of acute pleurisy, and the result of inflammatory processes.

(*b*) *Purulent Exudation.*—Empyema, the result of acute pleurisy. The quantity of pus may vary from a few ounces to many pints, and may then cause a bulging of the thoracic walls. The pus formed may be healthy, or it may be sero-purulent.

(*c*) *Hydrothorax.*—In this case the fluid is not the result of inflammatory action, but "a passive dropsy of the pleura." This condition generally co-exists with disease of the heart, causing transudation of fluid, due to an overloaded condition of the pleural veins, or with renal disease, causing hydræmia. The fluid is distinguished from that the result of pleurisy by the absence of coagulated fibrine, and the inflammatory changes in the pleural surfaces are wanting. Hydrothorax is also, as a rule, bilateral; pleurisy being unilateral.

(*d*) *Blood.*

The diagnostic signs of the presence of fluid in the thorax are, bulging of the side affected, dulness on percussion, if the quantity of the fluid be great, together with diaphragmatic breathing. The heart may be more or less displaced. The affected side during respiration is almost motionless, and the respiratory sounds are lost. Dulness on percussion varies with the position of the patient, chiefly, however, at the posterior and lower parts of the pleura. In some cases a splashing sound may be produced by suddenly moving the patient. In a case I have lately seen of thoracic effusion the patient, a man, astonished the bystanders by rolling from side to side to produce the splashing sound.

PNEUMONIA.

Lat. *Peripneumonia.* Fr. *Pneumonia.* Ger. *Lungenentzündung.* Syn. *Pneumonie.*

DEFINITION.—Acute inflammation of the parenchyma, or substance of the lung.

Pneumonia may be produced by a sudden chill, exposure to cold and wet, direct irritation or injury to the chest; it also often occurs secondarily to some acute affections, as measles, small-pox, typhus, typhoid, etc., and after surgical operations. Pneumonia is now considered by many as an acute specific fever, with local manifestations in the lungs. The disease is ushered in by symptoms which denote acute inflammatory action. A single, severe rigor, followed by nausea, pain in the side, quick pulse, sometimes reaching from 120 to 150 beats a-minute, together with thirst, and increase in the temperature of the body, mark the invasion. Pneumonia may be divided into two varieties :—

1. Simple, or *lobar*, or *croupous* pneumonia, where the whole lung or a lobe is affected.
2. *Lobular*, where distinct and scattered portions or lobules of the lungs are attacked. This, in most cases, occurs as a secondary affection in the course of pyæmia, or the fevers, bronchitis, etc.

It has become of late the fashion to apply the term *croupous* to that form of pneumonia known of old by the single word pneumonia, and for lobular pneumonia we now have *catarrhal pneumonia*, or perhaps better *broncho-pneumonia.* In the former, the exudation is said to partake of the character of the false membrane of croup; in the latter, of the nature of the bronchitis with which it is accompanied. Broncho-pneumonia is most common among children, especially during an attack of one or other of the eruptive fevers. It may also occur in the very aged, terminating a long chronic bronchitis. Pneumonia more frequently occurs in the right than in the left lung, but it may occur simultaneously in both, and is then spoken of as "double pneumonia."

An attack of pneumonia—by this we mean croupous pneumonia—may be divided into three stages :—

1. The stage of congestion or engorgement.
2. The stage of exudation or red hepatisation.
3. The stage of grey hepatisation or purulent infiltration.

The third—or more probably the *second*—stage in favourable cases is called the *Stage of Resolution.*

The *Stage of Congestion* is attended with symptomatic febrile movement, and pain in the side affected. The pain is, however, as a rule, slight, and is most marked when the pneumonia is complicated with pleurisy. The movement of the thoracic walls is diminished on the affected side. The vocal fremitus is sometimes, but not always, increased over the hepatised lung. There is cough and expectoration of a scanty, yellowish, transparent, viscid mucus. This soon, however, becomes semi-transparent, adhesive, and has a reddish tinge, not unlike that of iron rust; the *rusty sputa* of pneumonia, most marked in the second stage of the disease. The coloration is due to small quantities of blood, chiefly seen during the first stage, on the surface of the sputum, and not intimately mixed with it. Hurried breathing is one of the earliest symptoms of pneumonia. The patient generally lies on his back, or slightly inclined to one side. The temperature of the body may rise to 104° F.; a sudden increase in the temperature marking the invasion of another lobe of the lung. Percussion at first gives no sign of disease, but soon the percussion note is deadened and a sense of resistance is experienced by the finger. If auscultation be practised during this stage, a sound known as *minute crepitation*, or crepitant rhonchus, is heard over the affected lung. A bubbling noise is also heard at the end of each inspiration, and the breathing gradually becomes bronchial, this being more marked as the consolidation of the lung increases and is most audible in the posterior and lower regions of the thorax.

During this stage the following pathological changes are going on :—Intense hyperæmia, followed by a watery exudation, which soon solidifies. The lung is increased in size and weight; in fact, it has ceased to be full of air and is full of fluid, the change being as marked as when a dry sponge is suddenly plunged into water. It is highly probable that in the cases of recovery the disease has not extended beyond this stage, and that recovery becomes more and more problematical as the disease passes into succeeding stages.

Post-mortem Signs.—If at this stage death ensue the lung pits on pressure, and when cut into, will be found redder than natural; if it be also gently squeezed, or a knife scraped along the cut surface, a quantity of frothy reddish serum exudes. The tissue of the lung is soft and friable, thus distinguishing pneumonia from simple œdema of the lung.

If the disease be not arrested, the second stage, that of *Exudation*, *Solidification*, or *Red Hepatisation* ensues. The minute crepitation heard during the first stage disappears, and is replaced by bronchial respiration, bronchophony, and whispering bronchophony. There is dulness on percussion over the whole of the affected part. The sputum is now very tenacious, the patient finding some difficulty in removing it from his mouth. It is less ærated, more plentiful in quantity, and more deeply tinged with blood. As consolidation increases the sputum becomes less tough, is more easily brought up, and is more or less distinctly nummular, due to the greater abundance of pus corpuscles now present.

Post-mortem Signs.—The lung is solid, and when cut into resembles liver or spleen, its spongy structure being entirely lost. The air cells become entirely blocked up by the solidified fibrinous exudation, which may now be pressed from them, and which takes the place of the serum in the last stage. There is no evidence of any new formation of cells in this condition of the lung. The blood is stagnant in the vessels, or these may rupture and allow of extravasation more or less circumscribed.

The third and last stage is that of *Grey Hepatisation*, or *Purulent Infiltration.* During this stage all the normal breath sounds have disappeared, and we have, as in the latter part of the second stage, bronchophony and bronchial breathing. The temperature, which may have been marked by much variation, increasing as successive portions of the lung became diseased, may now vary from 102·2° to 104° F. When the temperature falls, the physical signs will indicate that the lung has become more or less consolidated.

The general symptoms are, gradually decreasing strength and increasing weakness. The chlorides are almost entirely absent from the urine. A herpetic eruption not unfrequently breaks out round the mouth during the course of the disease.

Delirium tremens is often a severe complication in the intemperate when attacked by pneumonia. Should the disease end in resolution, there is a gradual return to the normal condition of the organ. The favourable symptoms may set in suddenly or gradually, sometimes by profuse sweating, by bleeding at the nose, or by diarrhœa. The respiration diminishes in frequency, the pulse becomes slower, and the patient expresses himself as feeling better.

Post-mortem Signs.—The hepatised lung is now softer than in the second stage, and in old people is darker than in children, owing to the presence of more pigment. The lung is enlarged, very friable, and fills the cavity of the chest. When healthy, as is well known, the lung is found more or less compressed when the cavity of the thorax is laid open. Fatty degeneration may be also more or less present. The capillaries generally appear empty, which explains the grey colour of the lung. The exudation mass in this stage becoming detached from the walls of the air cells, gives the granular appearance to the organs. When "resolution" sets in, the mass becomes more granular and indistinct. This, the stage of "purulent infiltration," is the period when the contents of the air cells are prepared for removal by expectoration. Some of the infiltration is expectorated, the rest is absorbed. The physical signs of resolution are "redux crepitation," not unlike in character to the crepitation of the early stage, followed, after a period more or less prolonged, with the normal breath sounds.

The inflammatory cells poured out during the course of pneumonia are held by some to be the white blood cells, but mixed with these there must be also a large number of the red corpuscles. The pus formed is generally diffused through the pulmonary tissues—seldom circumscribed in an abscess.

Pneumonia may terminate in—

1. Resolution. 2. Gangrene. 3. Abscess.
4. Chronic Pneumonia. 5. Phthisis (?).

In some cases the pus may be absorbed, and resolution and recovery ensue; abscesses, however, may occur either diffused or encapsuled; the latter may then undergo caseous degeneration. Gangrene of the lung is exceedingly rare as a termination of pneumonia.

There are other forms of pneumonia, of which a brief mention will now be made.

CATARRHAL, LOBULAR, OR BRONCHO-PNEUMONIA.—This is the ordinary form of pneumonia occurring in young children, and is in most cases simply an extension of an existing capillary bronchitis. Broncho-pneumonia is perhaps most frequently met with during the course of measles. It differs from croupous pneumonia in the absence of the fibrinous exudation characteristic of that disease, the exudation in this being merely a proliferation of the cells lining the alveoli. Small portions of the lung are found after death in a hepatised condition, the air cells being filled with an exudation having its "origin, in part from a proliferation of endothelium, in part from the migration of cells from the blood vessels, and in part from secretion which has been sucked in from the bronchioles and plugged the alveoli of the lung-tissue." The lobules attacked may, after a time, lose their primary dark colour, and, becoming lighter, appear to the inexperienced as tubercles. The portions of the lung implicated may vary in size from that of a millet seed to a pigeon's egg. When the disease becomes chronic, the nodules of solid lung become disorganised and break down into cheesy masses. To this variety of pneumonia Niemeyer considered the greater number of cases of phthisis to be due.

Symptoms.—Those of bronchitis, which mark the onset of the disease. The temperature may rise to 102° or 104° F., and this sudden rise of temperature is an important symptom. The pulse is increased in frequency. The local signs are—increased difficulty of breathing, and short harsh cough, the sputum seldom becoming rusty.

The **Diagnosis** is more or less uncertain. There are no marked physical signs. Over the consolidated portions of the lung there may be increased vocal fremitus, and deficient resonance, etc. During an attack of broncho-pneumonia prominent brain symptoms may arise, almost leading one to suspect the presence of tubercular meningitis. The disease may run an acute or chronic course.

INTERSTITIAL PNEUMONIA.—Probably as one of the results of chronic inflammation, portions of the lung become indurated by the formation of a fibro-nucleated material. This material gives to the part of the lung affected a firm, dense, and hard feel, and is probably first formed in the walls of the alveoli, extending thence into the interlobular connective tissue. The natural pigment found in the lung is also increased, giving an iron-grey colour to the indurated parts. "The microscope shows a fibro-nucleated material, occupying nearly the whole of the pulmonary structures, although within the alveoli there may also be found cells and nuclei." The bronchial tubes become distended, and may be the seat of inflammatory processes which, extending into the portions of indurated lung, cause the tissue to break up and a cavity to be formed. By some the term "cirrhosis" has been applied to this diseased condition of the lung. "The chronic pneumonia or fibroid disease," says Wilks, "is never confined to one lung; it is, therefore, called phthisis, and has a constitutional origin, whereas the cirrhotic disease has often a mere accidental cause for its origin."

Symptoms.—This disease often runs a chronic course; the patient gradually emaciates, often complaining of uncomfortable sensations, or even pain in the chest, shortness of breath, etc. The physical signs are those of acute pneumonia becoming chronic. This condition of the lung is frequently combined with dilatation of the bronchi or bronchiectasis, which may produce during life all the physical signs of phthisical vomicæ.

The **Prognosis** in the several varieties of pneumonia will depend upon the extent of the lung involved, and the constitution of the patient. In most cases death takes place by asthenia, combined with apnœa, but more frequently by the former alone.

Treatment.—The usual methods adopted in the treatment of inflammation generally are applicable in pneumonia. Free purgation, bleeding in the robust and plethoric, hot poultices to the chest, and the administration of sudorifics and diuretics, will form the principal aids to recovery. Calomel may also be found useful. The application of four or six leeches to the chest will often afford intense relief, especially if morphia be subcutaneously injected after they have been removed. A pneumonia may often be arrested at its onset by a brisk purge, or by the wet pack, as recommended by Dr George Johnson. There can be no doubt that most of the cases of pneumonia result from a sudden chill, and, the blood being driven to the internal organs, congestion takes place. At this stage a hot bath, assisted by a dose of tartar emetic, will often cut short a pneumonia. Expectorants are not indicated during the earlier stage of the attack. Tartar emetic in small doses will be useful, if the inflammatory action be very great, and the patient strong and plethoric. Aconite has also been largely given in this disease. In catarrhal pneumonia, ipecacuanha and salines are useful. Attention should be paid to the past history of the patient; any hereditary tendency should be noticed. Pneumonia occurring in a patient of a gouty diathesis will often be relieved by the administration of colchicum, together with the appropriate medicines for pneumonia. During the second and third stages the general indication is to support the strength of the patient. Alcohol, tonics, and a nutritious diet will be found necessary, with great care and constant observation during convalescence.

TABLE SHOWING THE PHYSICAL SIGNS OF PNEUMONIA, EMPHYSEMA, AND PLEURITIC EFFUSION.

	PNEUMONIA.	EMPHYSEMA.	PLEURITIC EFFUSION.
Ascultation ...	Fine crepitation, bronchial breathing, and bronchophony.	Indistinct respiratory murmur, prolonged inspiratory and expiratory sounds. Dry crepitation.	The respiratory sounds are lost below the level of the fluid. They are at first bronchial.
Percussion	Dulness increasing as the disease proceeds.	Morbidly clear sound, and greater resonance than natural. The sound is not, however, tympanitic, as in pneumothorax.	Dulness in all postures when the effusion is very great. Displacement of the heart, and modification of its sounds. If the patient stand, there may be dulness only partial, greater low down where the fluid gravitates. The dulness supervenes more rapidly than in pneumonia. The transition from dull to healthy is very abrupt. Effusion also intercepts the slight fremitus, or vibration, which accompanies the voice, in all parts of the chest.

EMPHYSEMA.

Lat. *Emphysema.* Fr. *Emphyseme.* Ger. *Emphysem.*

Definition.—A condition of the lungs due in one case to abnormal dilatation of the air cells, in the other to infiltration of air into interlobular areolar tissue or sub-pleural tissue.

There are two forms of this affection, the one known as *interlobular* or *interstitial emphysema*, the other as *pulmonary* or *vesicular emphysema.* The former is due to the infiltration of air into the interlobular areolar tissue, or into the sub-pleural areolar tissue, the result of laceration or rupture of the air vesicles or bronchi from broken ribs, etc.; the latter to enlargement of the air cells, atrophy of their walls, and obliteration of their vessels, produced by any cause exerting a great strain upon them. The specific gravity of the portion of the lung affected is diminished; the lung becomes less crepitant, and feels doughy on pressure. The loss of elasticity prevents the collapse of the lung during expiration, and the vitiated air is not expelled; hence the want of oxygen, which may also be due to the diminution of the respiratory surface from one or more of the air cells being thrown into one. This is the form of the disease most frequently met with in practice.

Emphysema is usually slow in its development, and it may occur in the course of many lung affections, or of whooping-cough, croup, or heart affections, causing congestion of the pulmonary capillaries. Opinions vary as to the pathology of this disease; it has, however, been suggested by some that the plugging of a portion of the bronchial tubes, generally those leading to the posterior portions of the lung, by mucus in acute bronchitis, and subsequent collapse of the air cells so cut off, may account for the enlargement of the cells in connection with unobstructed tubes. This is the view advocated by Dr Gairdner, who maintains that emphysema occurs not during expiration, but during inspiration. Dr Gairdner does not state that this is altogether complementary, but that when one portion of the lung is functionally obstructed, another expands to supplement its bulk in the chest, not merely simply to make up for the loss of function of the diseased portion. This theory does not explain the fact that emphysema is most frequently met with along the anterior borders of the upper lobes and apices of the lungs on both sides. Hypertrophy of one lung when the functions of its fellow are impaired, must not be mistaken for true emphysema. The theory of Laennec, still maintained by some, was that during a bronchitis a tube became plugged; the air, however, during inspiration could enter, but could not escape during expiration, thus the lung gradually became distended. In opposition, however, to Laennec, it has been proved that the state of things he supposed as a cause for emphysema really leads to collapse of the lung. There is, however, evidence to show that during expiration, if there be any impediment to the breathing, portions of the lungs least subject to pressure are distended; and that although the plug theory of Laennec cannot be maintained, as first propounded, yet emphysema may occur during expiration, as shown by Sir W. Jenner.

The explanation is as follows:—If air be prevented from leaving the lung, the internal pressure in all parts will be increased, but it will only greatly distend those parts where the resistance of the thoracic walls is least, viz., at the apices and anterior portions of the lungs. Hence we have, as before-mentioned, emphysema most marked in the anterior borders of the upper lobes of the lung. Perhaps the safest way would be to say that probably no one cause is sufficient to produce the disease, especially in those cases where both lungs are affected, and where there is no history of bronchitis or mechanical obstruction, and where the emphysema appears to depend upon some obscure tissue changes.

Symptoms.—The symptoms which mark the pathological condition of emphysema are laboured respiration and dyspnœa, occurring in paroxysms. The sufferer is unable to indulge in any violent exercise. The surface of the body often presents the appearance of venous congestion, frequently well-marked in the cervical veins, owing to an accumulation of blood within the right cavities of the heart. According to Sir W. Jenner, the thorax becomes barrel-shaped, the sternum is arched, the antero-posterior, lateral, and vertical diameters are increased, the lower cervical, dorsal, and upper lumbar spine is curved backwards, the concavity forwards, the ribs are too horizontal, and the inter-costal spaces widened. In emphysema, the heart may be displaced downwards and to the right; whereas in effusion into the left pleura, if the amount of fluid is not large, simply downwards, the heart returning in most cases to its normal position when the fluid is removed and the lung expands.

The physical signs of this condition are, abnormal expansion of the chest, increased resonance on percussion, with weakened respiratory murmur, and sonorous râles.

The **Prognosis** is favourable, so far as the life of the individual is concerned, the disease being seldom fatal by itself; but it may materially incapacitate a man for performing his business occupations.

Treatment.—The treatment resolves itself into the treatment of the bronchitis, with which it is usually associated, and attempts to shorten the paroxysms when they occur. Smoking stramonium or weak tobacco may be tried. The administration of ether is also useful. The iodide of potassium in some cases possesses a marvellous effect, and should always be tried. A mountain residence may be recommended, as the more rarefied the air breathed the better for the comfort of the patient. Rarefied air allows the air vesicle to contract and thus diminishes the dyspnœa.

PNEUMOTHORAX.

Lat. *Pneumothorax.* Fr. *Pneumothorax.* Ger. *Pneumothorax.*

Definition.—An accumulation of air in the cavity of the pleura.

This disease may be due—1. To an opening in the pulmonary or parietal pleura admitting air. 2. Air may be secreted. Penetrating wounds of the thoracic walls, or the rupture of a vomica, are the chief causes which give rise to this condition. When due to the rupture of a vomica, or small phthisical abscess, death may suddenly follow the escape of air into the thorax.

Symptoms.—There may be deep-seated pain with dyspnœa, due to pressure on the lung by the air in the pleural sac. The side affected is not enlarged unless there be pyo-pneumothorax as well. The signs by which the presence of air in the pleural cavities may be detected are, a morbidly clear tympanitic or a metallic sound on percussion, and almost total loss of the respiratory murmur on the side affected. *Amphoric resonance* accompanies the breathing, and there is a metallic ringing sound with the voice and cough. The respiratory murmur on the sound side is increased in distinctness. Pneumothorax may be complicated with hydro-thorax; a splashing sound will be heard on succession when this is the case.

PHTHISIS.

Lat. *Phthisis.* Fr. *Tuberculeux.* Ger. *Phthisis.*

Definition.—A disease of the lungs which is characterised by progressive consolidation of the pulmonary texture, and by the subsequent softening and disintegration of the consolidated tissue.

I know of no disease of which it is so difficult to give a clear account as the one under consideration. To none can the old adage "*Tot homines quot sententiæ,*" be more correctly applied. Leaving out the "bacillus theory," to which, I think, the Scotch verdict of "not proven" may be justly applied, I shall try to give as clear a view as possible of the various opinions of this disease as are at present currently held.

In passing, and confining myself to pulmonary phthisis, I believe that phthisis is essentially an inflammatory disease occurring in a tissue of a low vitality due to some obscure hereditary conditions. Expose a dozen persons to like conditions and some will develope different kinds of pulmonary affections, bronchitis, pneumonia, pleurisy, etc., from which they will recover. Yet some will be attacked by phthisis and die. The healthy organs of the first recover themselves, those of the others break down under the strain. The inflammatory action in the latter is low in type, the inflammatory products are not removed, they tend further to degenerate, and by their presence increase the local irritation and further the disintegration and death of the surrounding tissues.

Pulmonary tuberculosis, or pulmonary consumption, consists in a deposit in the lungs of a morbid product, called tubercle, which undergoes the several phases of deposit, suppuration, ulceration, and discharge. The deposit of tubercle in the lung is but the local expression of a special condition of the system, known as the tuberculous cachexia, already described. (See "Tuberculosis," page 172.)

The above is a short epitome of the older teaching on this subjuct ; and it will at once be seen that *tuberculosis* and *pulmonary consumption* were controvertible terms. Now the doctrine held by some, and in this, as I have just said, I concur, is, that phthisis is essentially an inflammatory disease, that inflammation occurring in the lungs of debilitated persons, or those suffering from a scrofulous taint, results in the vesicles becoming plugged with inflammatory matter, and that unless this is removed in the ordinary way by resolution it undergoes certain fatty metamorphoses, and breaks up into a cheesy mass, which destroys the lung. "As regards miliary tubercles, Addison said that they were sometimes present and sometimes not. They had no direct histological relation with the softer material which he called inflammatory, and others yellow tubercle; but their presence merely showed the proneness of those who had them to consumption. Such persons were apt to have tubercles form in the parenchyma of the lung, and those same persons were liable to a low form of pneumonia, which not terminating in resolution, but in destruction of the lung, made them the subjects of phthisis." (Wilks' *Lect. Path. Anat.*) Niemeyer strongly maintained that the destructive material in the lung had an inflammatory origin, the early stages being a lobular pneumonia. Miliary tubercle, according to this authority, is an accidental growth. It is also now considered to owe its miliary or millet seed-like appearance to the rigid circumscription of the inflammatory process which is essential to its production. Tubercles are sometimes congenital and inherited, but are more frequently deposited during youth, in those of a tuberculous or scrofulous diathesis, induced by vitiated air, unhealthy occupations, and a poor diet. "More strictly speaking, it is not *disease* which is transmitted, but organs of such imperfect structure that they are liable to be thrown into a morbid condition by causes which sound organs could easily resist." (Combe's *Constitution of Man.*) The age at which phthisis generally makes its appearance is from eighteen to twenty-five.

Structural Changes of the Lungs in Phthisis.

1. An accumulation of epithelial cells within the pulmonary alveoli.	The material found here is composed of catarrhal products. The alveolar walls may be little if at all affected, but the contents of the alveoli may be found in all stages of retrogressive metamorphosis. As the disease advances, the alveolar walls are destroyed, all traces of structure lost, and nothing but granular *debris* left. The consolidated tissue is soft and friable, and may have a lobulated outline. Cavities of various sizes may be formed by the breaking down of the consolidated tissue.

2. The presence, within the alveoli, of a fibrinous exudation and leucocytes.	Exudation like that of ordinary pneumonia. The microscopical characters of the lungs like those of red and grey hepatisation.
3. A thickening of the alveolar walls by a small-celled tissue, together with, in most cases, the growth of a similar tissue around the terminal bronchioles.	The new tissue varies from an adenoid growth to one of denser structure. The pulmonary capillaries are entirely obliterated, and the lungs in part indurated. Softened yellowish caseous material now makes its appearance.
4. An increase in the interlobular connective tissue.	This becomes dense and fibrous, and causes the lung to contract. Large portions of the healthy lung tissue may be replaced by it.

The above Table is condensed from Dr Green's excellent work on Pathology and Morbid Anatomy.

Pulmonary consumption has been divided into *acute* or galloping consumption, and *chronic;* the former is a very rare affection. When it does occur the invasion is sudden, the symptoms are from the first greatly aggravated; hectic fever with markedly high temperature, profuse sweating, and diarrhœa rapidly follow, accompanied with rapid and persistent emaciation, death taking place in from three to twelve weeks, from exhaustion, or the extension of the tuberculous affection to the membranes of the brain. After death the lungs are found from top to bottom impregnated with miliary tubercle like the eruption of an exanthem, death having taken place before there is any considerable degeneration or softening.

Symptoms.—The early symptoms of phthisis are obscure; dyspepsia, loss of appetite, increasing dislike for fatty foods, slight, dry cough, more troublesome at night, and dyspnœa on exertion, fugitive pains in the chest, *loss of flesh, with concurrent loss of weight,* debility, pallor of the face and flattening of the chest, and profuse sweating during the night, which becomes so abundant that the patient's night-dress may be quite damp in the morning; hectic flushes on the cheek, quickening of the pulse towards evening, disturbed sleep. *Fever* is a marked symptom of phthisis, and its presence or absence by the aid of the thermometer night and morning should not be neglected. In women, the catamenia may be arrested. Diarrhœa occurs in a large proportion of cases. Sooner or latter an attack of hæmoptysis occurs; the patient is alarmed and seeks advice.

On examination of the chest during the first stage of the disease, slight dulness on percussion is found upon and below one clavicle, especially as compared with the other side. Loss of elasticity in the thoracic walls, and commencing immobility of the infra-clavicular regions of the chest. Tubercles are first deposited in the apex of the lung, particularly of the left side; and that side will be flatter than the other, and will not rise so freely during inspiration. The view held by some as to the "elective affinity" of tubercule for the upper lobes is that these parts are the driest, so that cacation of catarrhal products is more likely to occur there than elsewhere, and also that there is less expansion of the apices during breathing, thus admitting of accumulation in them. The pressure is less on these parts during coughing, which also tends to prevent the expectoration of the exudation from the air cells. Dr Moxon held that the relative activity of the upper lobes in respiration might account for their primary attack.. At any rate, the disease, as a rule, extends from the apices downwards. May not the modern method of wearing clothes account for the frequency of the attack in these parts. From two inches above the nipples to the neck, woman is relatively less clad than she is downwards from them, and the same holds good among men. The large sleeve holes and open portion of the waistcoat being noticed.

On auscultation, it will be found that the expiratory murmur is prolonged, and the following respiratory sounds may be detected, varying with the stages at which the patient first comes under notice. *Broncho-vesicular*, denoting moderate or slight solidification; *bronchial*, considerable or complete solidification, condensed lung being a good conductor of sound; and *cavernous*, the presence of cavities. If the hands be placed on the side of the chest, thoracic vibration will be felt when the patient is speaking. The voice, as time goes on, becomes weak and hollow, the dyspnœa greater, and the pain in the chest is increased, and the cough more and more distressing. As the disease proceeds, suppuration and ulceration take place in the substance of the lung, and a vomica or cavity is formed, which at length opens into a bronchial tube, and its contents are expectorated. The cavity is lined by a false membrane that continues to secrete pus. It is considered probable by some that vomicæ may heal by contraction of their walls, but this must be of exceedingly rare occurrence from the constant expansion of the chest in respiration. The expectoration now consists of pus and tubercular matter combined, having a distinct straw colour, and sinking in water. A gurgling sound, combined with large crepitation is now heard over the cavity, followed by a clear sound of the voice, called *pectoriloquy*, as if the patient were actually speaking in your ear. To produce this sound, the vomica must be empty, near the surface of the lung, and freely communicating with a bronchial tube. The cavity must not be too large, for over a large vomica there is heard a hollow sound of the voice, and cough and respiration known as *cavernous* or *amphoric* resonance. It is from an old vomica that the sputa most peculiar to phthisis comes, called *nummular sputa*, consisting of circular greenish purulent masses, that remain distinct in the vessels in which they are spat. What is the cracked-pot sound, or *Bruit de Pot fêlé* of Laennec? It is a sound produced by the sudden escape of air subjected to pressure, and in phthisis the following conditions are necessary for its production:—1. A moderate sized cavity close to the surface of the lung, and situated under a yielding portion of the chest wall. 2. Communication with a moderate sized bronchus. When the thorax between the first and the fourth rib, the usual situation of this sound, is suddenly struck, the air is forced out from the cavity along the bronchus, and the sound produced. It may be imitated by striking the closed hands on the knee. In health the sound may be produced in crying children, or adults when singing, and in disease it is most clearly heard if the mouth of the patient be opened. The sound may disappear for a time, owing to the communicating bronchus being closed with mucus, or the cavity emptied of air by repeated examinations. The febrile movement increases, and may assume an intermittent type, buoying up the patient with false hopes on the days the fever remits. The appetite is entirely lost, the sufferer refusing to be tempted with the nicest delicacies. There may be intense thirst and colliquative diarrhœa. The emaciation is now intense, the hectic flush is brighter; in some cases delirium supervenes, and the patient may die from hæmorrhage, a large vessel having become ruptured during an act of coughing, a gush of blood choking the patient—a state of things horrible to witness. Sometimes death takes place as it were during sleep.

Hæmoptysis is most frequent in the early stage of phthisis, when the presence of the tubercles first excites congestion of the adjacent parts of the lung. It is then in very small quantities, coming from ruptured capillaries. In the latter stages of the disease, when it may prove almost instantly fatal, it proceeds from some large vessel either ruptured or ulcerated in a vomica. Fatal hæmorrhage would be far more frequent were it not for the fact that the arteries are usually secured by a plug of coagulum before the ulcerative process sets in. Vomicæ very seldom open into the pleural cavity; when they do, there is immediate and severe dyspnœa, from air entering the pleural cavity and compressing the rest of the lung. This condition of things is known as *pneumothorax*.

Tubercles may be deposited during the course of consumption in almost every organ and structure of the body. Not unfrequently during the course of pulmonary phthisis the larynx becomes affected, and laryngeal phthisis is the result. Ulcerations form in the larynx, and the voice is lost.

Bulbous enlargement of the tips of the fingers, together with a curving in of the nails, due probably to malnutrition of the matrix of the nail, has been frequently noticed in the course of phthisis.

Treatment.—Whether or not the doctrine of the inflammatory nature of phthisis be the correct one, at any rate it is a good working hypothesis, and points to the necessity for protecting those who are known to have an hereditary tendency to lung affections from cold, and all the chances of setting up inflammatory action in the lungs. Apart from these considerations, there are three chief indications in the treatment of phthisis:—

1. To support and improve the patient's condition.
2. To obviate congestion of the lungs.
3. To soothe the cough.

The first indication is answered by a generous and nutritious diet, and the exhibition of cod-liver oil. Exercise in the open air, a strict regard to hygienic measures, together with tonic medicines, quinine, hypophosphates of soda, and lime, combined with gentian or calumba; the mineral acids, will greatly tend, if not to cure, at least to prevent the rapid course of the disease. The patient should be clothed in flannel, and if possible, during the winter, a residence at Bournemouth is advisable. In the early stages small blisters may be applied to the walls of the chest, or the thorax may pe painted with iodine. A stimulating liniment may also be used. The profuse sweating may be relieved by small doses of the sulphate of zinc, combined with digitalis, in a pill. The hypodermic injection of gr. $\frac{1}{100}$ of sulphate of atropia has been found useful in checking the profuse sweating in phthisis. To reduce the high fever in acute phthisis, Niemeyer's powder may be given, F. 4[illegible] The arsenical solution in three-drop doses, with the liquid extract of ergot, are useful when there is any hæmoptysis. For severe hæmoptysis Dr Dobell's mixture will be found of the greatest service, F. 12. Tepid sponging of the body with vinegar and water is very gratifying to the patient, and may soothe him to sleep.

The second and third indications will be best answered by residence in a moderately warm climate; the warm air soothes the bronchial tubes, and prevents internal congestions by keeping the blood on the surface. This is the opinion of some practitioners, but I think that the weight of evidence is in favour of a cool dry climate for phthisical patients; and it is wonderful how some of those unfortunates thrive by wintering in Canada or in protected stations on the Alps. Bamff, a town at the foot of the "Rockies," in the North-West Territories of Canada, is likely to become a favourite winter health resort for consumptives. The scenery is said to be very grand, and there are important thermal springs in the town. Protect the skin with flannel and send the patient out to take the air. Use a little common sense in the treatment of your patient; do not load the stomach with cough mixtures, tonic mixtures, etc. Mixtures containing morphia, phosphoric acid, or the hydrocyanic acid, with some sweet demulcent, may be given, if necessary.

CANCER OF THE LUNG.

Lat. *Carcinoma pulmonalis.* Fr. *Cancéreux pulmonaire.* Ger. *Krebs der Lunge.*

Carcinoma of the lung is a very rare affection. When it does occur as a primary disease, it is usually of the medullary or encephaloid variety; and then usually begins in the bronchial glands, thence extending along the bronchial tubes into the parenchyma of the lung. It must be distinguished from pulmonary tuberculosis and chronic pneumonia by the absence of the symptoms which betoken these diseases.

HÆMORRHAGES OF THE LUNG.

Under this head three varieties of pulmonary hæmorrhage will be mentioned.

1. Hæmorrhage into the bronchi and finer air tubes as a result of *congestion* variously produced, phthisis, cancer, etc.

2. Hæmorrhagic infarction, due to the plugging of a branch of the pulmonary artery by an embolus escaping from the heart or a systemic vein. In either case the clot may be absorbed or remain as a fibrous knot, which may subsequently undergo caseation or produce a "metastatic abscess."

3. Pulmonary apoplexy. This is a condition of the lung which, in most cases, may be said to be complementary to long-standing heart disease, chiefly of the mitral valve. There is extravasation of blood into the air cells of the lung, and sometimes an infiltration of the interstitial tissue. In fatal cases the blood may be either found coagulated in the air cells forming solid masses, varying in size from a pea to a walnut, the nodules feeling like hard projecting tumours, or the blood may be more widely diffused, and larger portions of the lungs are solidified. The lungs sink in water. The term "splenization" is sometimes given to this condition of the lung. Pigmentation may occur, known as "brown induration," and if inflammatory products are also thrown out, we may get the so-called "brown indurated pneumonia." The vessels of the lung may become varicose and atheromatous.

The **Diagnosis** of these conditions is attended with some difficulty, but hæmoptysis in varying quantity occurs in nearly all cases of pulmonary hæmorrhage. The respirations are hurried and laboured, and *percussion* will probably yield dulness over more or less of the lung. A crepitant ronchi due to blood in the air cells and smaller air tubes will be heard on auscultation. There is an absence of fever. Examine the heart.

The following Table gives the characteristic sputa in the undermentioned diseases :—

Pneumonia.—*First Stage* : *Engorgement.*—Scanty and tough, chiefly mucus with no definite shape, containing a large quantity of air, and floating as a spumous layer on water. When blood is present it is in minute quantities, generally on the surface of the sputum, not intimately mixed with it. This latter character is diagnostic of pneumonia before the physical signs of the disease are developed. *Second Stage: Hepatisation* —Sputum copious in quantity, less ærated, very tenacious, and deeply tinged with blood, which is *intimately* mixed with the mucous, and gives it the characteristic *rusty* colour, different from the streaky appearance of the sputum of the first stage. Fibrinous clots, casts of the finer bronchi, may now be seen on tilting up the receiving glass, or more clearly brought into prominence by washing the sputum, when they appear of a nearly pure white colour. *Third Stage: Resolution.*—The sputum becomes yellowish, less tenacious, not adherring to the sides of the vessel; more opaque, due to increase of pus cells, disappearance or fatty degeneration of the fibrinous clots of second stage, gradual return to a pure bronchial secretion, and ultimate cessation of all discharge. If resolution does not take place, sputum becomes fluid, frothy, reddish-brown—*prune juice sputum;* if abscess forms, the sputum becomes greenish-yellow and purulent; if gangrene, fetid dirty greyish colour, with shreds of dead lung tissue.

Broncho-Pneumonia.—The sputum of ordinary bronchitis, being simply muco-purulent, with only slight and transient traces of blood.

Phthisis.—Acute miliary tuberculosis, sputum of simple bronchitis. Chronic cheesy degeneration before excavation, sputum of ordinary bronchitis. Elastic fibres sometimes seen with the microscope. When a cavity is formed, the sputa become firm, rounded, or *nummular*, opaque, yellowish-green or greyish colour, containing little air and sinking in water. The sputa may be mixed with freshly effused blood, and more or less bronchial secretion. The sputum of gangrene of the lung and the putrid stage of bronchiectasis differs from that of caseous pneumonia and pulmonary abscess by containing few or no elastic fibres.

Bronchiectasis.—The sputum is brought up at long intervals, and then in considerable quantity, of a yellowish-green colour and offensive odour, said to resemble that of a soap factory.

Bronchitis.—First, simply mucus, frothy, glairy, vitreous, and transparent; later on in the disease, the sputa become pellet-shaped, irregular globular masses of a dull yellowish colour, muco-purulent. In miner's phthisis, or rather chronic bronchitis, the sputa may be charged with black pigment.

INFLAMMATORY AFFECTIONS OF THE HEART.

PERICARDITIS.

Lat. *Pericarditis.* Fr. *Péricardite.* Ger. *Pericarditis.*
Syn. *Entzündung des Herzbeutels.*

Definition.—Inflammation of the pericardium, attended with more or less effusion of serum into the pericardial cavity.

Pericarditis may be divided into three stages: the first is the period of invasion, and ends when effusion has taken place in sufficient quantity to be appreciated by physical signs; the second is the stage of effusion; and the third that of recovery.

Exclusive of any traumatic origin, pericarditis is most frequently a secondary affection, often occurring during the course of acute rheumatism, in Bright's disease of the kidneys, in the eruptive fevers, in pleurisy, in pneumonia, in pyæmia, and in scurvy, and in the last has been described as "hæmorrhagic pericarditis."

Symptoms.—The development of the inflammatory action is attended with more or less pain in the precordial region; sometimes the pain is acute and lancinating, not unlike the pain in pleurisy, for which disease, at this stage, it may be mistaken. If the patient be carefully watched it will be noticed that he holds his chest as immovable as possible, and especially restrains the action of the diaphragm. To do this more effectually patients have been known to tie broad belts tightly round their bodies. At other times there may be very little, if any, pain; and attention may scarcely be drawn to the condition of the heart till effusion, the result of the inflammatory action, has taken place. An irritable suppressed cough is often present, accompanied with some dyspnœa. The countenance of the patient expresses great anxiety and distress. The pulse is quickened, becomes smaller and harder, and sometimes irregular and intermittent. Pain is experienced on pressure in the region of the heart, or under the cartilages of the ribs. If the disease be not now arrested, the symptoms become aggravated; there is pain on swallowing, explained by the fact that the œsophagus for a part of its course is in contact with the pericardium. The action of the heart is tumultuous, the fever is high, and great anxiety is expressed by the patient, who also complains of inability to lie on the left side. The nervous derangement is greatly increased, and a peculiar form of delirium, sometimes quiet, at other times noisy, is not unfrequent. Auscultation in the first stage detects the "to and fro" sound, due to the rubbing together of the dry inflamed surfaces of the pericardium, best heard just above the left nipple or under the sternum. This soon ceases, either from the presence of effusion, or the gluing together of the opposite surfaces of the pericardium. When effusion has taken place, the area of cardiac dulness is increased and altered, becoming pyramidal in shape, the base being downwards and the apex pointing upwards; the dulness also extends further to the left than the apex beat of the heart, a diagnostic sign of great value. There may even be bulging of the chest walls if the effusion is large in quantity. The heart's impulse is displaced, and altered in force and rhythm, the apex beat being suppressed when the effusion is large, but on placing the patient on his back the apex beat may again become appreciable, and the percussion sound clearer. The proper heart-sounds also become muffled and weakened, and when the pericardium becomes adherent to the heart a double second sound is heard. When this is present the area of dulness is not much, if at all, increased, and pericarditis, with exudation of lymph, is present. Should the heart become adherent to the pericardium and this to the walls of the thorax, the intercostal spaces will be depressed during each systole, but such depressions are not distinctive of mere adhesion of the heart to the pericardium. When these depressions exist, the apex of the heart is *not* *usually* felt to impinge against the side, the reason for this being, that the organ is confined under the sternum by its unnatural attachments. The thoracic depression will of course be more marked if the pericardium be also adherent to the spinal column. If endocarditis be also present, a loud systolic bellows-murmur will be heard, indicating the presence of fibrinous deposit in and on the valves of the heart.

Pathology and Morbid Anatomy.—In the earliest stage the pericardium is found injected, but soon a layer of lymph is formed which spreads over the surface of the heart, extending from the origin of the great vessels. If effusion into the pericardium does not occur, the heart becomes glued to its investing membrane. In some cases the solid lymph has a honeycombed appearance, due to the presence of effusion in the interstices of the lymph. When the effusion is present in moderate quantity, the lymph-coated surfaces of the heart and pericardium, by their constant rubbing together, become rasp-like, and cause that peculiar rough sound, differing from the slight "to and fro" sound heard in the early stage of the disease. Sometimes, due to the separation of the adherent pericardium by fluid, ragged portions of lymph are found floating in the effusion, or attached to the surfaces of the heart or pericardium. In debilitated or strumous persons the effusion may become purulent, the quantity present amounting to thirty ounces or more. The disease may terminate by resolution, the effusion of lymph and fluid becoming absorbed. In less favourable cases, after the lapse of some years, the heart undergoes atrophy, and is diminished in size.

Diagnosis and Prognosis.—So various and so liable to modification are the symptoms of pericarditis, that considerable care is required in the diagnosis of the disease. Dr Latham mentions two cases in which the disease was mistaken for disease of the brain and treated as such. Inflammation of some other thoracic viscus may be readily mistaken for it; but it must be borne in mind that the most latent cases of pericarditis have occurred in patients whose thoracic viscera were in other respects sound. The "to and fro" sound before mentioned as an early sign of inflammation of the pericardium, occurs earliest and most frequently at the base of the heart, but it may be absent in some cases, as it may also be present in others, although not the result of inflammation. A friction sound may be associated with peculiar spots on the surface of the heart, known as "milk spots," about the nature and production of which opinions differ. They are probably the result of friction. Pericardial murmurs may be known by their being not permanently synchronous with the systole or diastole, but occurring irregularly in the cardiac area; they may also be caused to disappear at one part and reappear at another, by changing the attitude of the patient. Dulness, on percussing the region of the heart, which increases from day to day, is also a diagnostic sign of some importance. The prognosis should be guarded; pericarditis, however, rarely kills in its acute stage, but effects are frequently produced which result in early death. It may, however, prove fatal in the acute stage, when the effusion is considerable, from pressure on the auricles causing death by asphyxia.

Treatment.—The treatment of pericarditis includes also the treatment of the diseases with which it may be associated; and this fact should be borne in mind, for the treatment applicable in acute rheumatism would be obviously dangerous when the pericarditis occurs in typhus, typhoid, etc. When it occurs in acute rheumatism, very little is necessary beyond the usual methods of treatment adopted for that disease, together with the application of belladonna liniment or a belladonna plaster to the precordial region. Sometimes a few leeches may be applied over the region of the heart, at other times linseed meal poultices will be found very useful. Saline purgatives, with a restricted diet, are indicated in the first stage if the disease is a primary affection, which it very rarely is. Aconite may also be given during this stage with marked results Opium is invaluable, and should be given in sufficient doses to relieve the general and local symptoms. Blisters are not, as a rule, required at the outset, although I have seen the application of a blister followed by immense relief to the patient. The patient may also, in some cases, be brought early under the influence of mercury with considerable advantage. During the second stage —that of effusion—the removal of the effused fluid is called for. The application of small blisters, or painting the precordial region with iodine, together with the administration of cathartics and diuretics, will best meet the requirements of the case. During the third stage any undue excitement must be avoided, and measures taken to strengthen the system and the action of the heart. If there is reason to believe that the fluid is purulent, or if its pressure cause urgent symptoms, paracentesis of the pericardium may be necessary.

TABLE GIVING THE DIAGNOSIS OF ENDOCARDIAL MURMURS FROM THE FRICTION SOUNDS OF PERICARDITIS.

ENDOCARDIAL MURMURS.	FRICTION SOUNDS.
1. The bruit is soft and blowing.	1. Different shades of rubbing, grating, and creaking.
2. Usually single and synchronous with the pulse and impulse.	2. Usually double ; louder during the diastole than systole.
3. Prolonged so as in some cases to obscure the natural first sound.	3. Short ; seldom obscures the valvular sounds.
4. Murmur distant and deep-seated.	4. Superficial and near.
5. Murmur not limited to the precordial region.	5. Sound limited to the precordial region.
6. Murmur persists in every stage of the disease, and in every posture of the patient.	6. Varies in intensity at intervals ; is modified by posture ; disappears more quickly under treatment.

ENDOCARDITIS.

Lat. *Endocarditis.* Fr. *Endocardite.* Ger. *Endocarditis.*

DEFINITION.—Inflammation of the membrane lining the cavities of the heart and valves.

Endocarditis is almost exclusively limited to the left side of the heart, and is then generally found attacking the valves of those parts most exposed to friction. Like pericarditis, it is in most cases associated with other diseases. Apart from the kind of endocarditis just mentioned, some writers describe another form under the name of *ulcerative endocarditis*, characterised by the formation of fungoid vegetations and ulcerations of the endocardium. The onset of the disease is sudden, accompanied with rigors, high fever (104° to 108° F.) of an intermittent or remittent type, tenderness and enlargement of the spleen and other organs, probably due to infarctions of decomposing material separated from the cardiac ulcerations. In some cases the symptoms assume a typhoid or pyæmic character. In the cardiac vegetations Micrococci are frequently found, more probably a result than a cause of the disease. Death generally ensues in a few days.

The diagnosis from simple acute endocarditis is not always easy, the history of the case and the severity of the symptoms must be our guide. It must be borne in mind that in simple endocarditis embolic infarctions are comparatively rare, and tenderness of other organs equally so.

The disease may be mistaken for typhoid fever, ague, and pyæmia. There can, however, be little excuse for such a mistake, if the symptoms peculiar to the diseases mentioned are taken into consideration. Pyæmia may follow ulcerative endocarditis, or the latter may cause the former; the question, however, is of minor importance, as the sufferers from either die.

Symptoms.—The symptoms of endocarditis are not unlike those which accompany pericarditis, with which it is frequently associated. On auscultation a murmur is detected like that heard in cases of chronic valvular disease. The murmur is usually of a soft, bellows character, of mitral origin, and accompanying the first sound of the heart, being heard loudest at or near the apex of the heart. If during the course of an attack of rheumatic fever, a murmur occurs which was not present at the outset, endocarditis may be diagnosed. In auscultating a supposed case of endocarditis, we have to decide whether the abnormal sound is endocardial or exocardial; is the murmur old or recent, is it mitral or aortic? Of chronic endocarditis it may be said that an accelerated and somewhat embarrassed state of respiration is one of its most constant symptoms. Even when at rest the patient complains of his "difficulty of breathing," and on the slightest exertion or mental excitement this difficulty becomes intensified.

Endocarditis is not likely to prove fatal in the acute stage, but its after effects are more serious than those of pericarditis, because the valves never recover their healthy condition.

Pathology and Morbid Anatomy.—Infiltration takes place in the deeper layers of the endocardium, and young cells are quickly produced. The new tissue thus formed projects and forms minute granulations and vegetations upon the surface of the valves. After death there is little sign of inflammatory redness in the valves. The endocardium may ulcerate, but most frequently the morbid condition of the membrane undergoes fatty or calcareous degeneration. Hypertrophy and dilatation of the heart often ensue, the patient dying prematurely of dropsy. Sometimes the little nodules of fibrine on the valves—the product of the inflammatory action—become detached, and entering the circulation are carried and lodged in some distant artery, as one of the cerebral arteries, and thus cutting off the supply of blood, lead to mal-nutrition, softening of the brain, apoplexy, paralysis, and aphasia. The plugging of one of the arteries of the extremities may occur, and gangrene of the limb ensue. This condition is known as embolism.

Treatment.—For the most part the treatment recommended to be adopted in pericarditis is applicable in this disease. The administration of alkalies is indicated in the hope that they may reduce the excess of fibrine, or render it more fluid, and thus prevent its deposit on the valves of the heart. Mercury may be given so as to bring the system under its influence, and in many cases this course is attended with the happiest results. In rheumatic endocarditis, salicin in preference to the salicyl compounds is recommended by some, as the latter appears to have no power of controlling or arresting the cardiac complications when once they have developed themselves.

MYOCARDITIS.

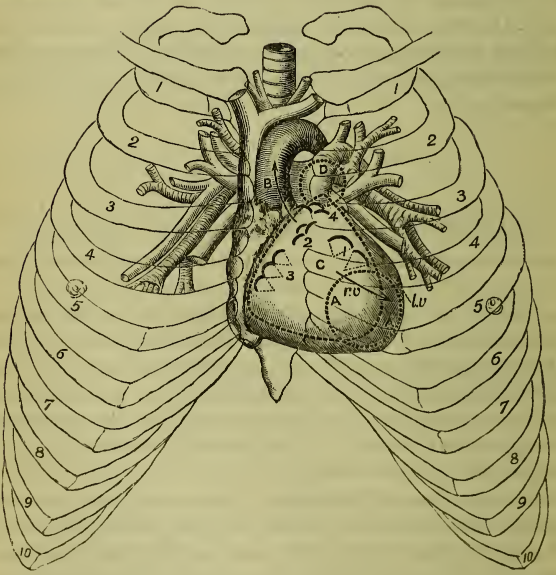
Lat. *Myocarditis.* Fr. *Myocardite.* Ger. *Myocarditis.*

Definition.—Inflammation of the muscular structure of the heart.

This is an extremely rare affection, consisting in inflammation of the muscular structure of the heart. The disease may arise in three ways: extension of inflammation from the pericardium, or endocardium, or the inflammatory action may begin in the substance of the heart itself. It is generally associated with pericarditis or endocarditis.

In some cases of rheumatic fever, which have ended fatally, the muscular tissue of the heart is found swollen, softened, opaque, and slightly mottled, pus being easily scraped from its surface with the dissecting knife. Ulcers may form on the surface of the heart; these are generally due to an ulcerative process beginning at the valves of the heart, caused by excessive inflammatory action. From the valves the ulceration extends to the muscular tissue which is found hanging in jagged pieces in the cavity of the ulcer, and which, becoming detached, are swept into the general circulation, where they end in plugging some artery, probably in the brain, too small to allow their passage. The left ventricle is that part of the heart most frequently affected.

Suppuration may take place, the pus being discharged into the pericardium, or into one of the cavities of the organ. The abscesses formed are probably of pyæmic origin. The presence of myocarditis cannot be detected during life.



A. The seat of the mitral murmurs. B. Seat of the aortic. C. Seat of the tricuspid. D. Seat of the pulmonary. *r.v.* Right ventricle. *l.v.* Left ventricle. 1. Mitral valves. 2. Aortic. 3. Tricuspid. 4. Pulmonary.

The 'arrows' point to the direction of the murmurs in aortic and mitral disease.

STRUCTURAL DISEASES OF THE HEART.

ANATOMICAL RELATIONS OF THE HEART.

In our examination of the heart, and indeed of the thorax generally, we should not omit the four sources of knowledge—*inspection*, *palpation*, *percussion*, and *auscultation*. The first informs us of increase of size of the precordial region; the second leads us to recognise the intensity of the pulsations; the third enables us to decide as to the size of the heart, from the area of dulness or condition of the lungs in its vicinity; and the fourth helps to distinguish normal from abnormal sounds in the precordial region.

In practising auscultation over the heart care must be taken not to press too heavily on the precordial region, as in thin subjects, and in women and children, a murmur, generally pulmonic, may be produced by the pressure.

The heart is so situated in the thorax that its axis is directed from above, downwards, forwards, and to the left—two-thirds of its bulk being to the left of the median line of the sternum, and one-third to the right. The left auricle, the highest point of the heart, is found just under a line joining the lower edges of the sternal cartilages of the right and left second ribs, the apex lies just behind the sixth left costal cartilage to the inside of the mammillary line. Draw a curved line with the convexity outwards from the second intercostal cartilage to the apex of the heart; this will give the left boundary of the organ. The greatest convexity of the curve must not be more than three and a half inches from the messial line. Now, draw a second curved line from a point about half-an-inch from the sternum in the second right intercostal space to the sternal insertion of the fifth rib. This line, distant at its greatest curve about one and a quarter inches from the right border of the sternum, marks the right boundary of the heart. Join the lower ends of both curves, and the line uniting them gives the lower boundary of the heart. From the second to the fourth rib the heart is covered by the lungs, only a little more than half of the right ventricle and the apex being uncovered. The area of *absolute* or *superficial* dulness corresponds to that portion of the heart free from the lungs, the area of *relative* dulness to that covered by lung. The *upper* limit of the absolute cardiac dulness begins, as a rule, at the *upper border of the fourth left rib;* the *left* coincides with a slightly curved line passing from the superior border of the fourth rib to the apex of the heart, *inside* the mammillary line; the *right* is marked by the *left* edge of the sternum from the *fourth* to the *sixth* rib.

VALVULAR DISEASES OF THE HEART.

In the great majority of cases, the valves of the left side of the heart are those most frequently affected, viz., the aortic and mitral.

The appearances which the valves present after death vary greatly. In some cases they may be merely thickened and contracted; in others, they may be covered with vegetations differing in size. They may also be the seat of atheromatous or calcareous deposits.

If auscultation be practised over the region of the heart, two well-marked sounds, following each other in rapid succession, succeeded by a pause, may be distinctly heard.

To understand the valvular diseases of the heart it is necessary to know the position of the valves and the normal sounds of the heart. The mitral valves are between the left auricle and ventricle, the tricuspid valves between the right auricle and ventricle. The *first* sound is caused by the closing of these valves during the ventricular systole, assisted by the ventricular muscular contraction, shown by—

1. Presence of sound in extensive disease of mitral valve.

2. May become indistinct, with mitral valve healthy, when muscular structure of heart diseased in myocarditis or fatty degeneration.

3. Sound longer than can be accounted for by transient tension of auricular valves.

That the first sound is not entirely due to muscular action is shown by—

1. *Absence* of systolic sound at apex when the aortic valves are to a high degree incompetent. The loss of the sound is due to absence of full tension of mitral valves, a result of the blood flowing into the ventricle through the insufficiency of the aortic valve, and thus lessening the difference between the *initial* and *final* tension of the mitral valve.

2. Presence of sound modified but present, due to tricuspid valve being intact, which, as before shown, helps in the formation of first sound.

The semilunar valves are between the pulmonary artery and right ventricle. The aortic valves between aorta and left ventricle. The *second* heart sound is due to tension of these valves by backward pressure of blood during the ventricular diastole.

The *first* sound is dull and prolonged, heard with the greatest intensity at the apex—that is, just under the fifth rib and fifth intercostal space; precedes the pulse at the wrist, and coincides with the impulse of the heart—that is, with the systole of the ventricles.

Reduplication of the *first sound* may occur in healthy persons. The first sound, as just shown, is chiefly caused by the closure of the mitral and tricuspid valves. Any cause which prevents the simultaneous closing of the valves will produce a double sound; in other words, if the two factors in the production of the first sound be separated, a reduplication or "doubling" of the normal sound will result. The reduplication is therefore supposed to be due to one or all of the following causes:—

1. Non-simulantaneous contraction of the two ventricles, leading to irregularity in the tension of the mitral and tricuspid valves.

2. Non-synchronous tension of the individual segments of the auriculo-ventricular valves.

3. Tension of the arterial walls during systole, later in point of time than the tension of the auriculo-ventricular valve.

Slow action of the heart is necessary to enable us to distinctly hear the double sound.

The *second* is a shorter and sharper sound, following close upon the arterial pulse, and succeeded by a period of silence. It is heard most distinctly over the situation of the aortic and pulmonary valves, at about the level of the third costal cartilage, and is coincident with the diastole of the ventricles. If the time of a cardiac pulsation be divided into ten parts, the first sound will take up four-tenths, followed by a short pause of about one-tenth; the second sound two-tenths, and the succeeding silence or long pause three-tenths.

Reduplication of the *second sound* occurs most often in *mitral stenosis*, and is held to be due to—

1. Non-coincidence of the closure of the aortic and pulmonary valves, resulting from unusual difference in the quantity of blood contained by the aorta, as compared with that in the pulmonary artery.

2. Due to the narrowing of the mitral orifice, hence removed by increasing the action of the heart, a loud murmur resulting. When the pericardium becomes adherent to the heart a double second sound may sometimes be heard.

The normal sounds of the heart may be variously modified by disease, and we may have murmurs arising from changes within the heart, *endocardial;* and from changes outside the heart, *pericardial.* Endocardial murmurs are produced by—

(*a*) Anatomical changes in the valves or valvular orifices, giving rise to incompetence of the valves or constriction of the orifices. In each case there is some obstruction of the circulation.

(*b*) Deposits on the endocardial lining of the ventricles, etc., which, however, offer no impediment to the passage of the blood.—*Organic Murmurs.*

(*c*) Want of uniformity in the tension of the valves and arterial walls, the structure of the heart being normal.

(*d*) Dilatation of the cardiac end of the aorta, a normal aortic orifice being present.—*Inorganic Murmurs.*

(*e*) Murmurs are also produced when a fluid passes into another fluid with a velocity less than that of the incoming current. Thus, in aortic stenosis the blood rushing from the left ventricle with a greater velocity than that of the blood in the aorta, produces a murmur. Sounds are produced by uniform, murmurs by non-uniform, vibration. Murmurs are primarily pertaining to fluid, probably intensified by the roughness of the valves, or diseased condition of the lining membrane of the heart. Murmurs due to actual impediment to the circulation are called *organic;* when no such cause is present, *inorganic.* The arterial murmurs may be due to the transmission of cardiac murmurs, to local causes in the arteries, carotid, etc., or to simple pressure as in the superficial arteries in the limbs.

The signs by which disease of the *aortic* and *mitral valves* may be detected, are thus tabulated:—

Bruit:—If *systolic* and loudest at	*Base*—Aortic *obstruction.* *Apex*—Mitral *insufficiency.*	
Bruit:—If *diastolic* and loudest at	*Base*—Aortic *insufficiency.* *Apex*—Mitral *obstruction.*	
Pulse:—If *regular*	Full or strong Jerking, resilient	Aortic disease.
Pulse:—If *irregular*	Intermittent, unequal Soft, small, weak	Mitral disease.

A frequent pulse, with regularity or irregularity, but without confusion or tumult, betokens nervous or inorganic derangement.

Two results follow disease of the valves: contraction of the orifice or dilatation, and consequent inefficiency of the valves. The one results in *valvular obstruction*, or *stenosis;* the other in *valvular insufficiency*, or *regurgitant disease of valves.*

The mitral valve, when diseased, gives rise to the following phenomena:—a systolic and a diastolic or pre-systolic murmur, heard best towards the apex of the heart on the left side. The former, or systolic, more or less replacing the first cardiac sound, is caused by the regurgitation of the blood from the ventricle into the auricle; the latter, diastolic, or pre-systolic, as it occurs at the latter end of the long cardiac pause and just before the ventricular systole, is due to the impediment to the passage of the blood from the auricle to the ventricle.

Of all the valvular lesions, mitral regurgitant is the most common. As a portion of the blood that ought to go into the aorta is sent back into the auricle, we have anæmia in front of the valve, and engorgement behind; thus regurgitation gives rise to cough, dyspnœa, and pulmonary congestion, also to hypertrophy of the left and right ventricles, and dilatation of the left auricle. After a time, however, the ventricles may become dilated, due to some inherent disease in their muscular structure, or to causes resulting from the primary disease of the valve. As a result, also, of mitral incompetency, we may get tricuspid incompetency. The liver, spleen, and other organs become, to a more or less extent, affected, and general dropsy ends the scene. The pulse becomes irregular, and unequal in force and fulness.

Disease of the aortic valve is attended with a systolic and a diastolic murmur.

The systolic murmur occurs when the blood is prevented from freely flowing out of the ventricle, and is heard most distinctly at or about the base of the heart; it is generally propagated along the course of the carotids. A systolic aortic murmur may occur with a normal orifice, if the aorta be dilated at its origin.

The diastolic-aortic or aortic-regurgitant murmur heard at the base of the heart, near the left margin of the sternum, represents insufficiency of the aortic valve, and regurgitation from the aorta into the left ventricle.

Disease of this valve does not produce pulmonary or general systematic congestion, unless associated with hypertrophy of the ventricle, and subsequent disease of the mitral valve.

The pulse is generally abrupt, short, and jerky, and it is in this form of valvular disease that we meet with the "water hammer" or Corrigan's pulse.

Lesions of the tricuspid valve are rare, and are, as a rule, secondary to disease in other valves. Tricuspid regurgitant murmur occurs with the first sound, and is to be distinguished from *mitral regurgitant* by its being heard about the right inferior border of the heart, near the ensiform cartilage. Tricuspid regurgitation is accompanied with *true venous pulsation in the neck*, best seen in the external jugular vein on the right side. The venous pulsation in the jugular during inspiration and expiration is at once distinguished from a true venous pulse by its rhythm.

Pulmonic murmurs due to organic disease are extremely rare; when present, they are to be detected near the left margin of the sternum, in the second intercostal space.

Disease of the *left* side of the heart alters the force and character of the pulse. Disease of the *right* side causes obstruction to the circulation, venous congestion, and œdema.

The symptoms which first draw attention to the state of the heart are slowly increasing dyspnœa and palpitation of the heart, increased on ascending hills or stairs.

An œdematous condition of the ankles and eyelids may also be present. The physical sign is a distinct and permanent murmur or *bruit*, modifying one or more of the natural sounds of the heart. The dyspnœa is peculiar, being rather a *breathlessness* than a difficulty of breathing. A form of dyspnœa, especially seen where the right heart is dilated and fatty, has had the term "Cheyne Stoke's respiration" applied to it. For a few respirations the patient experiences considerable difficulty of breathing. The dyspnœa then gradually subsides, the respirations becoming shallower and shallower, slower and slower, when they may for half to three-quarters of a minute be entirely suspended. They then gradually reappear, and become deeper and deeper, quicker and quicker, until the height of the paroxysm of dyspnœa is reached. The duration of the respiratory cycle from the height of one paroxysm to that of the next may extend over two minutes.

Although not characteristic of any one form of organic heart lesion, the "Cheyne Stoke's respiration" is a symptom of grave and ominous import, presaging, as a rule, a speedily fatal termination. The cause of this form of respiration has not as yet been satisfactorily explained. Some believe that in consequence of the deficient supply of oxygen, the irritation of the respiratory centre is so lowered that a much larger accumulation of carbonic acid in the blood is required to excite it into action.

Murmurs may be sometimes only functional, no organic lesion being present. These *inorganic* murmurs, as they are called, are known—

1. By their softness, short duration, and feebleness, and by the absence of harshness, sawing, or rasping.
2. They are *never diastolic*, but as a rule systolic.
3. Most frequently heard at the pulmonary orifice, next at mitral orifice.
4. Generally in chlorotic anæmic persons combined with venous murmurs in the neck.

5. They are only occasional and temporary, thus differing from the permanent murmurs of valvular disease.

The subsequent evils attendant on valvular disease are congestion of the internal organs, dilatation and hypertrophy of the cavities of the heart, and dropsy more or less diffused.

Prognosis.—The prognosis will depend upon the condition of the heart, and the power it may have to carry on the circulation.

Treatment.—The treatment may be considered under two heads:—*First*, to avoid excessive action of the heart; and, *second*, to treat the complications as they arise.

The first indication is carried out by the avoidance of excessive muscular exertion, and the abuse of alcoholic stimulants, which increase the rapidity of the circulation.

The second indication is best met by the exhibition of digitalis, hydrocyanic acid, belladonna, and such remedies as appear to possess the power of relieving the irregularity of the heart's action without diminishing its power. When dropsy occurs, diuretics, if the kidneys are healthy, and hydragogue cathartics may be employed. The administration of ether, or dry cupping over the region of the heart, will often relieve the dyspnœa. In mitral disease, digitalis is most useful, and it may be combined with iron. In aortic lesions, belladonna is often of great value, and is preferred to digitalis by many practitioners.

TABLE SHOWING THE PHYSICAL SIGNS AND RESULTS OF DISEASE OF THE VALVES OF THE HEART.

	NATURE OF DISEASE.	POSITION OF MURMURS, ETC.	CHARACTER OF PULSE.	DISEASES PRODUCED.	GENERAL SYMPTOMS.
PHYSICAL SIGNS SHOWING	Disease of the *right* auriculo-ventricular orifice, obstructing the onward passage of the blood. *Tricuspid obstruction or stenosis.*	So rare as not to need detailed description misleading to the student.			
	Disease of the *right* auriculo-ventricular orifice, permitting regurgitation of the blood from the ventricle into the auricle. *Tricuspid regurgitant or insufficiency.*	Maximum intensity of murmur over lower part of sternum, just above the ensiform cartilage. Scarcely audible at the apex of the heart. Not heard in the back or at the base. It is systolic.		Passive congestions, dropsy, when insufficiency is complicated with mitral obstruction. Nearly always complicated with aortic and mitral obstruction.	A small, irregular, intermittent pulse, perceptible in the jugular veins; called venous pulse. Characteristic of disease of the right side of the heart.
	Disease of the *left* auriculo-ventricular orifice, obstructing	Maximum of intensity above the left apex; between the cartilages of the fourth	Pulse small, *irregular*, intermittent, with a	Pulmonary congestion, dyspnœa, difficulty	Precordial beat, quivering, tremulous motion,

PHYSICAL SIGNS SHOWING	the onward passage of the blood. *Mitral obstructive or mitral stenosis.*	and fifth ribs. It occurs during the auricular systole. The murmur is therefore diastolic or presystolic. When there is insufficiency as well stenosis, the diastolic murmur is combined with one that is systolic in rhythm.	confused and bounding throb.	in breathing, dilated jugular veins, venous pulsation, cyanosis and anasarca, hypertrophy and dilatation of the right ventricle. Frequently sudden death.	coinciding with second sound. Murmur, that of a saw and file rolling into end of second sound, and obscuring the presystolic sound. Prolonged towards the apex, where it is most intense.
	Disease of the *left* auriculo-ventricular orifice, permitting regurgitation of the blood from the ventricle to the auricle. *Mitral regurgitant or insufficiency.*	Maximum intensity above left apex drowning the natural first sound of the heart. It is systolic. Sometimes audible at the inferior angle of left scapula and left axilla. Faintly heard at the ensiform cartilage and base of the heart.	Pulse small irregular, intermittent, with a peculiar tremulous action.	Congestion of lungs, kidneys, and liver.	The left auricle first dilated and hypertrophied, then the right ventricle and tricuspid regurgitation.
	Disease of the pulmonary orifice, obstructing the entrance of blood. *Pulmonic obstructive or stenosis.*	Maximum intensity over third left cartilage, close to the sternum. It is systolic. Cannot be heard at back. Heard along left edge of sternum; inaudible at level		Dilatation and hypertrophy of right ventricle and auricle.	

TABLE—*Continued.*

TABLE SHOWING THE PHYSICAL SIGNS, ETC.—*Continued.*

	NATURE OF DISEASE.	POSITION OF MURMURS, ETC.	CHARACTER OF PULSE.	DISEASES PRODUCED.	GENERAL SYMPTOMS.
PHYSICAL SIGNS SHOWING		of the first interspace. Not transmitted towards apex or along the aorta. Very rare.			
	Disease of the pulmonary orifice, preventing its closure. *Pulmonic regurgitant or insufficiency.*	Diastolic murmur heard over second left intercostal space.		Dilatation and hypertrophy of the right ventricle.	
	Disease of the orifice of the aorta, obstructing the entrance of blood. *Aortic obstructive or stenosis.*	Heard over the middle of the sternum on a level with the third right rib; inaudible, or nearly so, at apex. Systolic. Synchronous with pulse at wrist and impulse of the heart. Audible at the manubrium sterni, and sometimes transmitted into carotid, subclavian, etc. Heard also clearly at second right "aortic" cartilage, slightly over left or "pulmonic" cartilage.	Pulse small, soft, slow, and regular. If hypertrophy be present, pulse intermittent.	Palpitations, dyspnœa, sometimes blueness of the face, local pain, dropsy more or less intense, and more slow in appearing; œdema, local then general. Venous pulse rare.	Hypertrophy of left ventricle, mitral regurgitation may follow, due to extension of disease to mitral orifice or forcing of the valves from blood pressure.

PHYSICAL SIGNS SHOWING	Disease of the orifice of the aorta preventing its closure. *Aortic regurgitant or insufficiency.*	A diastolic murmur heard over middle of sternum, level with lower border of third right rib. Synchronous with the ventricular diastole. Propagated downward along the sternum, and frequently heard most clearly over ensiform cartilage.	Pulse large, full, regular, undulating or bounding, perceptible in the course of the principal arteries. Corrigan's or the "water-hammer" pulse present.	Palpitations, throbbing in the region of the heart, singing and swimming in the head, giddiness, sleeplessness, œdema towards the end of the disease; sometimes spitting of blood. Sudden death.	Hypertrophy and dilatation of left ventricle. An intermittent murmur heard in the crural artery.

Note.—Valvular disease of right side of the heart is rare. The most common murmurs are *mitral regurgitation*, *aortic obstruction*, and *aortic regurgitation.*

The following is the relative frequency of these murmurs:—

1. Mitral regurgitant.
2. Aortic obstructive.
3. Aortic regurgitant.
4. Mitral obstructive.
5. Pulmonic obstructive.
6. Tricuspid regurgitant.

The following combination may also be present:—

1. { Mitral regurgitant.
   { Mitral obstructive.
2. { Aortic obstructive.
   { Aortic regurgitant.
3. { Mitral regurgitant.
   { Aortic regurgitant.
   { Aortic obstructive.

HYPERTROPHY OF THE HEART.

Enlargement of the heart may take place without valvular lesion.

Hypertrophy is a thickening of the muscles of the heart, either by a growth of the fibres already existing, or more probably by the addition of new ones, and occurs chiefly in the ventricles, especially of the left side.

Dilatation is enlargement of the cavities of the heart, and occurs chiefly on the right side, being in all probability an idiopathic disease.

Hypertrophy *without dilatation* is known as simple *hypertrophy;* when they are combined, as eccentric *hypertrophy*, or *hypertrophy with dilatation;* and when there is hypertrophy *with lessening in the capacity of the organ*, as concentric.

"These terms," says Dr Wilks, "may be kept in mind, since they are useful in examinations; but they are for the most part of exceedingly little importance, and must have arisen in some author's study rather than in the course of inspection of the heart after death."

Dr Wilks, in opposition to Rokitansky, Bamberger, and some others, denies the existence of concentric hypertrophy; and if we understand him correctly, he considers this condition as showing a mode of death, the heart being in a state of systole, persistent after death—simple dilatation being the heart caught, as it were, in its diastole. Thus, if in a case of supposed concentric hypertrophy sufficient time be allowed, the heart will relax, and the case be described as one of simple dilatation; all morbidly dilated hearts being at the same time hypertrophied. Dilatation with thickening, and dilatation with thinning of the walls of the heart, are both cases of dilatation with hypertrophy; the only difference being, that in the one case the hypertrophy has exceeded the dilatation, in the other the dilatation the hypertrophy.

Causation.—These conditions arise from any cause that disorders or hampers the heart's action, such as valvular disease, aneurism of the aorta or of one of its first branches, or some functional cause, as long-continued over-exertion. It appears that soldiers, due to the absurd notions relating to the drill imposed upon them, suffer greatly from *irritability* and hypertrophy of the heart. The greater number of cases of *simple* hypertrophy occur along with granular disease of the kidney, and it is then chiefly limited to the *left* ventricle.

Symptoms.—The symptoms of hypertrophy are, constant excessive palpitation, dyspnœa, a full strong pulse, flushed face, headache, the pain being referred to the top and back of the head, and vertigo. The impulse of the heart is strong and heaving, but the sounds are indistinct, the first being so prolonged as to obscure the second.

The symptoms of dilatation are feebleness of the pulse and coolness of the surface of the body. If the right cavities are greatly dilated, general dropsy may ensue. The action of the heart is weak and undulatory, but the sounds of the heart are loud and noisy. "As soon as dilatation sets in, then begin the symptoms, and the trouble is in proportion to the dilatation. Simple hypertrophy does not cause symptoms, and never exists as a primary cardiac disease" *(Wilks).* In hypertrophy and dilatation the area of cardiac dulness is increased; when the left ventricle is enlarged, the increase is most marked in *length*, when the right, in *breadth.* But dulness *is not always present* in enlargement of the heart, as in cases where there is unusual over-lapping or thickness of the lungs in the precordial region without the lungs being necessarily diseased, and the left ventricle may be considerably hypertrophied without their being any increase in the area of normal dulness. In emphysema, for instance, the concomitant enlargement of the heart may be obscured.

Hypertrophy occurs in strong and young subjects, dilatation chiefly in the weak and feeble; and of the two, hypertrophy and dilatation, the latter is by far the more serious affection, the danger to life being in proportion as the dilatation exceeds the hypertrophy. Dilatation frequently arises from fatty degeneration of the muscular walls of the heart.

Treatment.—The treatment will consist chiefly in the application of general principles. It is important that rest be enjoined. When the hypertrophy is conservative, that is, when it is due to valvular disease, it does not require treatment. The excessive action of the heart may be moderated by aconite, a most valuable remedy, one or two minims of the tincture being given three times a-day, belladonna, veratrum viride, and hydrocyanic acid. Digitalis is contra-indicated except when the palpitation is marked showing the presence of commencing cardiac failure. Over-exertion should be avoided. In dilatation the treatment is the same, the chief object being directed to improve the tone and vigour of the muscular walls of the heart, and also to support the strength of the patient. Digitalis may in this case, if cautiously given, be of great use.

ATROPHY OF THE HEART.

Atrophy of the heart may be due to pressure exerted on the walls of the heart by the adhesions that follow pericarditis, or it may occur in connection with many exhausting diseases, especially cancer. The heart may decrease in weight from the normal standard—ten to twelve ounces in the male, or eight to ten in the female—to four and a-half ounces, and may become so reduced in size during the course of diabetes as to weigh only five and a-half ounces. Before pronouncing a heart to be atrophied, it should be remembered that the mode of death has much to do with the apparent smallness of the organ.

FATTY DEGENERATION OF THE HEART.

The deposit of fat within the muscular structure of the heart, and the gradual substitution of the former for the latter, constitute the disease known as Fatty Degeneration of the Heart. It differs from obesity of the heart in this, that the former is a degeneration of the muscular walls into fat, the latter is merely an accumulation of fat round the heart.

Fatty degeneration of the heart may occur singly, but it is generally associated with like conditions in other organs, as the liver and kidneys. A fatty condition of the cornea known as the *arcus senilis* is also often present, but as this state may occur without the heart being affected, it is chiefly important as a diagnostic sign of a constitutional tendency to fatty change, rather than of disease in any particular organ. When the heart is cut into after death, it is found to present a yellowish or fawn colour, the fibrous structure is almost lost, and its place is supplied by granules of fat, which give it a streaky, oily appearance. This appearance is now supposed to be due more to a re-arrangement of the muscular fibres of the heart than to large additions of fat.

Fatty degeneration may occur with or without valvular lesion, and may be general, or confined to one side of the heart only. It should be borne in mind that it may also occur in lean subjects.

Symptoms.—The symptoms are feebleness of the heart's action, and a pulse often irregular, slow, and feeble. Paroxysms of dyspnœa often occur, accompanied with pain and a sensation of pressure in the region of the heart. Fatty degeneration is rare in males under fifty, and in females under forty. The details of an interesting case are perhaps of more value to the student than the mere enumeration of symptoms.

Some years ago, a lady, a friend of the writer, was attacked by disease in her right ovary, probably of an inflammatory nature. This incapacitated her from taking active exercise, and as she was in easy circumstances there was no stint in the good things of this life. Her general health was in nowise affected, and as her appetite was good, she lived perhaps too freely for one not able to take healthful exercise. As a result of her sedentary life, combined with her good living, she soon became the victim of fatty degeneration. For some few years this change must have been going on, till at last heart symptoms set in, and a consultation was proposed by her usual medical attendant. The services of an eminent physician were sought, who pronounced her case to be one of pericarditis, followed by hydropericardium. He stated that there was evidence of considerable effusion of fluid in the pericardium, and proposed the usual remedies for this state of things. To counteract the constant tendency to fainting and palpitation of the heart, from which the patient suffered, stimulants were somewhat freely allowed. This course of things continued for some time (twelve or eighteen months), the lady enjoying tolerable health during this time, interrupted by the fainting fits and palpitation above mentioned. At last the fainting fits became so frequent that recourse was had more often than before to the use of stimulants; and after an attack of the symptoms of a more aggravated character, recourse was again had to the physician who had previously seen her. On this occasion he pronounced the heart to be in a better condition than when last examined, and that all fluid had been absorbed, and expressed the opinion that the patient was then suffering from irritation of the stomach, due to the large amount of stimulants prescribed to keep her from fainting, or dying from exhaustion as was feared. Before leaving he gave a favourable prognosis of her case. Three or four days after this she died. At the request of her husband, the family medical man and myself made a *post-mortem*. We found at least four and a half inches of fat between the skin and abdominal muscles, which were the subject of far advanced fatty degeneration. I may state that the enlargement of the abdomen had been attributed to ovarian dropsy, and I believe that at one time the necessity for performing ovariotomy had been seriously discussed. On examining the right ovary, we found it shrivelled and hardened; the left was healthy. The other internal abdominal organs were all more or less undergoing fatty degeneration. The muscles of the thigh were also cream-coloured from the same cause. On examining the heart there was only a very small quantity of fluid in the pericardial sac, and not the slightest signs of old adhesions or traces of previous pericarditis, but the heart was flabby, and almost entirely devoid of healthy muscular tissue. It was soft and greasy, with here and there streaks of the natural muscular fibres of the organ.

Now, here we have a case full of the most valuable lessons. First, a diseased ovary incapacitating from healthy exercise; a too free diet for the sedentary life that was led by the patient; and lastly, the administration of stimulants under the impression that the patient must take them or die. The case was by no means an easy one to treat; but the practical rule to be laid to heart is this, that in all cases where a sedentary life is rendered necessary, a rigidly abstemious diet must be enjoined. The disease here did not affect the general health; it merely condemned the patient for a time to sit still, and to move about with circumspection, till, as in this case, a natural cure was effected. But while this cure was being brought about, the seeds of that fatty degeneration were sown which ultimately proved fatal.

Death may take place suddenly from rupture of the organ, as was the case with the late Dr Abercrombie; or by syncope, as in the case of Dr Chalmers. When rupture does take place, it generally occurs in the ventricle, the left more frequently than the right. An over-accumulation of blood in the ventricles, due to deficiency in the contractile power of the organ, is the immediate cause of death in some cases.

Treatment.—The treatment is more hygienic and dietetic than medicinal. All excitement must be avoided when the presence of this condition is suspected. The diet should be nourishing, but limited in quantity and quality, and should consist chiefly of animal food deprived of fat. Exercise in the open air should be insisted upon. Tonics and medicines, to relieve the urgent symptoms as they arise, will often tend to prolong life in comparative comfort.

CYANOSIS.

Lat. *Cyanosis.* Fr. *Cyanose.* Ger. *Cyanose.* Syn. *Blausucht.*

DEFINITION.—A malformation of the heart, accompanied with a blueness of the skin, due to the circulation of arterial and venous blood mixed.

A malformation of the heart originating in early fœtal life, and very frequently due to an attack of endocarditis affecting the pulmonary valves, gives rise to cyanosis. The malformation in question is thus produced: owing to the more or less perfect closure of the pulmonary valves, the ventricular septum, which ultimately divides the heart into two cavities, is arrested in its development, and the blood freely mixing in the heart, cyanosis is produced. This affection was formerly attributed to an open foramen ovale or ductus arteriosus, but such an opinion Dr Wilks declares to be unfounded, and states that the most frequent cause of cyanosis is the malformation above mentioned. An obstructed pulmonary artery, according to Wilks, "necessitates all these further changes; a passage of blood into the left ventricle, and then a way out again into the pulmonary arteries by the fœtal ductus arteriosus, remaining patent. All these different alterations in the heart would so clearly arise from an obstructed pulmonary artery, that we think there can be little doubt that here lies the *fons et origo mali.* In the three or four cases of malformation of the heart in persons who have grown to adult age, the peculiarities have been of the kind we mention." In some cases the aorta is placed over both ventricles, and the foramen ovale remains open between the auricles, but if the obstruction to the flow of blood be compensated for in any way the auricles are relieved, and the foramen closes as usual.

In a disease thus depending on a malformation of an organ, no treatment is available.

FUNCTIONAL DISORDERS.

Functional disorders of the heart consist in a disturbed action of the organ without any organic lesion.

Severe palpitation occurring on the slightest excitement, or without any assignable cause, and accompanied with intense excitement on the part of the patient, who lives in daily dread of sudden death, are some of the characteristic symptoms of these affections. Sometimes the patient will complain of intense pain over the region of the heart, and also of inability to lie on the left side. Persons suffering from these derangements are most persistent in the belief that their hearts are irrecoverably diseased, and will go from one medical man to another in the hope of having their surmises confirmed. They suffer pain and palpitation of the heart, and "it must be diseased." Your arguments against their opinion are of little avail.

These disorders of the heart are mainly due to errors in diet, to an anæmic condition of the blood, to the abuse of alcoholic stimulants and tobacco, or to excessive sexual intercourse and self-abuse. Those of a gouty diathesis are often sufferers to a great extent from palpitation and other functional disorders of the heart.

The treatment consists in gaining the confidence of your patient and thus relieving his anxiety as to the severity of the disease, and in a careful removal of the cause.

THE FOLLOWING TABLE FROM AITKEN MAY BE OF USE:—

FUNCTIONAL DISORDERS OF THE HEART.

PALPITATION DEPENDING UPON ORGANIC DISEASE OF THE HEART.	PALPITATION INDEPENDENT OF ORGANIC DISEASE OF THE HEART.
1. More common in the male than the female.	1. More common in the female than the male.
2. Palpitation usually comes on slowly and gradually.	2. Palpitation usually sets in suddenly.
3. Palpitation constant, though more marked at one period than another.	3. Palpitation not constant, having perfect intermissions.
4. Impulse usually stronger than natural, sometimes remarkably increased, heaving, and prolonged; and at others, irregular and unequal.	4. Impulse neither heaving nor prolonged; often abrupt, knocking and circumscribed, and accompanied by a fluttering sensation in the præcordial region or epigastrium.
5. Percussion elicits a dull sound over an increased surface, and the degree of dulness is greater than natural.	5. Extent of surface in the region of the heart, which yields naturally a dull sound on percussion, not increased.
6. Palpitation, often accompanied by the auscultatory signs of diseased valves.	6. Auscultatory signs of diseased valves absent; *bruit de soufflet* often present in the large arteries, and a continuous murmur in the veins.
7. Rhythm of the heart regular, irregular, or intermittent, its action not necessarily quickened.	7. Rhythm of heart usually regular, sometimes intermittent; its action generally more rapid than natural.
8. Palpitation not much complained of by the patient, occasionally attended by severe pain, extending to the left shoulder and arm.	8. Palpitation often much complained of by the patient; readily induced by mental emotion; and frequently accompanied by pain in the left side.
9. Lips and cheek often livid; countenance congested; anasarca of lower extremities common.	9. Lips and cheeks never livid; countenance often chlorotic; anasarca absent, except in extreme cases.
10. Palpitation increased by exercise, by stimulants, by tonics, etc.; relieved by rest, and frequently also by local or general bleeding, and an antiphlogistic regimen.	10. Palpitation increased by sedentary occupations, by local and general bleeding, etc.; relieved by moderate exercise, and by stimulants or tonics, particularly the preparations of iron.

ANGINA PECTORIS.

Lat. *Angina pectoris.* Fr. *Angine de poitrine.*
Ger. *Angina pectoris.*

Definition.—A disease probably due to spasm of the muscles of the heart, resulting from extreme contraction of the systemic arteries.

Angina Pectoris is a disease of advanced life, occurring in paroxysms, attended with intense pain in the region of the heart, the pain extending down the left arm, and often amounting to indescribable anguish. The sufferer clutches at the nearest object for support. The palpitation of the heart is very distressing, accompanied with a feeling of faintness, and a dread of immediate death. The surface is cold and clammy, and great anxiety and suffering is expressed by the countenance of the patient. The attacks become more frequent, and in the course of a few weeks or months one proves fatal.

Some hold that angina pectoris is a form of neuralgia. Be this as it may, the most constant morbid appearances found after death are ossification of the coronary arteries, fatty degeneration of the heart, and valvular disease. Fortunately it is not a common disease.

Treatment.—The treatment is chiefly palliative, and consists in relieving the paroxysms by the administration of alcohol, ether, and other anti-spasmodics. The inhalation of nitrate of amyl, three drops on a handkerchief, is of value in severe cases. Nitrate of amyl acts by suddenly reducing the arterial tension or spasm, Should the arterial tension become habitually high, one drop of a one per cent. solution of nitro-glycerine in spirit may be given three times a day. The drug is particularly useful to prevent rather than to relieve the attack. During the intervals the general health of the patient should be improved; belladonna and the bromide of potassium may be given if medicines are employed, and a plaster of the former should be worn over the cardiac region.

EXOPHTHALMIC GOÎTRE.

Lat. *Exophthalmica vel morbus Basedowii, morbus Gravesii.*
Fr. *Goître exophthalmique.* Ger. *Basedowsche Krankheit.*

Definition.—A disease the result of unknown causes, characterised by increased action of the heart, enlargement of the thyroid gland and marked protuberance of the eyeballs.

It is not a litttle difficult to find a suitable location for this disease; in the nomenclature of the College of Physicians it is placed among the diseases of the thyroid gland, but it does not appear that this body has any particular connection with the disease as a cause. Some have suggested that disease of the cervical sympathetic ganglia producing irritation may be the prime cause, if not of the disease, at least of some of its symptoms. It is commonly known as *Graves'* or *Basedow's* disease.

Symptoms.—The disease more frequently occurring in women than in men, may come on suddenly, especially in highly nervous and anæmic persons. Sudden and severe grief or great anxiety are supposed to be among the exciting causes. In whatever way it may make its appearance, the chief cardinal points about the disease are the increased action of the heart, accompanied with exaggerated cardiac impulse, and of the sounds with a blowing systolic murmur. The cardiac area of dullness is not increased. Coincident with the increased action of the heart, or soon after, the thyroid gland is seen to enlarge, and the eyes become markedly protuberant; in some cases so marked is the protuberance that the eyelids cannot be made to cover the eyeballs. Besides the above, the patient is frequently irritable, suffers from sleeplessness, headache, loss of memory, disordered digestive functions, and emaciation. diarrhœa & pulsations

Prognosis.—The disease does not appear, as a rule, to end fatally.

Treatment.—Iron and iodine, if not injurious, do not appear to be of any use. Arsenic, ergot, digitalis, etc., may be tried. Galvinism to the sympathetic in the neck is reported to be of considerable use.

Belladonna & Ferrum -

THORACIC ANEURISM.

An aneurism may occur in any portion of the thoracic aorta, or in the great vessels within the chest. The usual seat of aortic aneurisms is the ascending portion of the arch outside the pericardium, but sometimes they occur within the pericardium, rupture sooner or later taking place, followed by sudden death. The tumours vary in size.

The author has a photograph of a poor man, a late patient of Edinburgh Royal Infirmary, who suffered from an aneurismal tumour of the thoracic aorta, so large that absorption of the sternum and upper costal cartilages took place. In the above case two-thirds of the aneurismal tumour were within the chest, the other third projecting externally. The age of the patient was thirty-eight. He died of asthenia.

Symptoms.—The symptoms will vary with the position and size of the tumour, and generally consist of those dependent on the pressure of the tumour on the various structures within the thorax. When small, they may not be detected during life. Dyspnœa occurs when the aneurism presses on the bronchi, trachea, larynx, or lungs, and the respiratory murmurs are greatly diminished. In those cases where the recurrent laryngeal nerve is subject to pressure from the aneurism, paralysis of the muscles takes place, and the breathing of the patient may become stridulous, and tracheotomy may be performed to relieve the symptoms if the true nature of the case be not suspected. When the aneurism is formed in the anterior wall of the ascending aorta the symptoms are less marked, and its growth may not be detected till it begins, as in the above-mentioned case, to show itself through the absorbed sternum and ribs. The dorsal vertebræ may be quite eroded, paraplegia being produced by the pressure on the spinal cord, for nothing seems able to resist the constant pulsating force of an aneurism. The arch of the aorta is the part most frequently attacked; and next, that portion of the thoracic aorta as it passes through the diaphragm. Auscultation will often detect a purring sound as the blood flows along the contracted artery, the maximum intensity of the murmur being most marked at the point which coincides with the murmur of aortic stenosis. But the position of the murmur varies with the part of the aorta affected.

1. *Ascending portion.*—Second right intercostal space and over the sternum.
2. *Transverse portion.*—Higher up and to the left of the sternum.

Hypertrophy of the left ventricle is not unfrequent in cases of aortic aneurism. Pressure of the aneurism on the œsophagus will be attended with pain and difficulty in swallowing. The general symptoms which follow any obstruction to the circulation will be present, and cause attention to be drawn to the state of the heart and vessels.

Treatment.—The treatment is only palliative. All mental or bodily excitement must be carefully guarded against, whilst the general health of the patient is improved. Mr Hulke has lately successfully treated a case of sub-clavian aneurism by starvation. He strongly recommends the treatment, and his arguments in its favour are to all appearance sound. Mr Tuffnell has also obtained good results from rest in the recumbent posture and a very carefully regulated diet. Among medicines the iodide of potassium would seem the best, and may be given in large doses.

DISEASES AFFECTING THE DIGESTIVE SYSTEM.

INFLAMMATION OF THE MOUTH.

Inflammation of the mouth or stomatitis is a disease of childhood. The following forms will be described:—

(*a*) Simple erythema.
(*b*) Diphtheria of the mouth.
(*c*) Follicular stomatitis.
(*d*) Vesicular stomatitis.
(*e*) Cancrum oris, or gangrenous stomatitis.

(*a*) Simple Erythema is often due to teething, gastric derangement, or to the mechanical action of hot or irritating substances taken into the mouth. The child is first noticed to refuse the breast from the pain of sucking, and then attention is drawn to the mouth.

Treatment.—The treatment consists in removing the cause, and correcting any gastric derangement when present.

(*b*) Diphtheria of the mouth may occur at any age, but is chiefly a disease of infancy. The mouth becomes covered with a diphtheritic exudation, with here and there small irregular patches, which are renewed as fast as they are thrown off. There is more or less general derangement of the system, and pain is felt on attempting to swallow. Fatal diarrhœa may also occur.

Treatment.—By way of treatment the mouth should be painted over with a solution of the chlorate of potash in glycerine, or a little borax may be sprinkled into the mouth. Saline mixtures may be given if necessary.

(*c*) Follicular Stomatitis.—This occurs chiefly in delicate children, and may cause considerable alarm among parents, who are always sure that nothing less than diphtheria is present. There may be some slight feverish symptoms, but, as a rule, the application of the chlorate of potash, as in the last case, soon removes the disease. The chlorate of potash is also almost a specific in *ulcerative stomatitis*, a disease more or less connected with some constitutional disturbance.

(*d*) VESICULAR STOMATITIS, APHTHÆ, or THRUSH, consists in the presence on the lining menbrane of the mouth and the tongue of small, round, greyish vesicles, which burst and discharge a clear watery fluid. It is most frequently a disease of infancy, especially in children of a weakly constitution, and not unfrequently follows attacks of measles, scarlet fever, or any other debilitating disease. It may occur in adults during the course of other diseases, and is then frequently the forerunner of a fatal issue. The writer has seen cases of an aphthous condition of the mouth, accompanied with a similar condition of the vagina, occurring in adults; and in one case, that of a pregnant woman, it was complicated with abortion. Thrush is often attended with the production of a microscopical fungoid growth *(oïidium albicans)*, but it is not quite clear what part the fungus plays in the causation of the disease, for thrush may occur without the presence of the parasite being detected.

Treatment.—The treatment consists in the use of mild alteratives and tonics, and washing the mouth out with a solution of the chlorate of potash or sulphite of soda.

(*e*) CANCRUM ORIS, GANGRENOUS STOMATITIS, or NOMA.—This disease occurs in childhood, and may lead to considerable annoyance to the medical attendant, who may be accused of salivating the child. It generally ends fatally, occurring most frequently after severe scarlet fever, typhoid fever, measles, etc. It first appears on the cheek as a pale-red patch, with slight swelling; then a slough forms, which may go on extending till the inner surface of the cheek becomes a foul smelling and painful ulcer. The disease may commence on the gums. The constitutional disturbance is considerable. The pathology of the disease is obscure; some vague connection with fatty degeneration of the liver has been suggested.

Symptoms.—Difficulty in mastication; extreme fœtid smell of the breath, the odour being characteristic of mortification; increased salivation, the saliva dribbling from the mouth; the countenance is anxious, and the glands of the neck swollen and hard. There is always more or less fever. This disease must not be confounded with syphilitic stomatitis. The history of the case, and the age of the patient, will assist the diagnosis.

Treatment.—Application of the nitrate of silver, or of strong nitric acid, to the sore; washing the mouth out with a solution of chlorate of potash, chloride of zinc, or chlorinated soda. Tonics, bark, quinine, the mineral acids, and strong beef-tea, milk, and in some cases brandy, may be administered in small doses.

TONSILLITIS.

Lat. *Inflammatio tonsillarum.* Fr. *Amydalite.*
Ger. *Entzündung der Mandeln.*

Definition.—Inflammation of the tonsils.

Cynanche Tonsillaris. Esquinancie. Angina Tonsillaris.

Tonsillitis, inflammation of the tonsils, or quinsy, is an acute inflammatory affection of one or both tonsils, often terminating in suppuration, and the formation of an abscess within the gland, which ultimately bursts, discharging its contents into the mouth.

The relief to the patient when this occurs is intense and immediate.

Symptoms.—Slight febrile symptoms, with a sensation of tenderness at the back of the throat, usher in the attack. Deglutition is performed with difficulty, often attended with intense suffering. Sometimes the febrile movement is very severe, and the temperature may vary from 102° to 105° F.; but when the danger to life from suffocation appears imminent, the abscess in the tonsil bursts, and the patient is relieved at once, though remaining weak for some time afterwards.

Sometimes the inflammatory action will suddenly leave the throat to appear as an acute attack of inflamed piles. When both tonsils are affected, the patient complains of a sensation of suffocation, he expresses great anxiety as to his condition, and may become delirious.

Tonsillitis may be due to cold or to sudden chills; in some cases the tendency to the disease is hereditary, and it would appear to be very frequently connected with the gouty diathesis. Every attack renders a future one more probable. Sometimes after repeated attacks of inflammation, the tonsils remain chronically enlarged, and should then be removed with the knife.

Treatment.—The treatment consists in the local abstraction of blood in the early stage of the disease, by making punctures into the tonsils with a sharp-pointed knife. If the disease be not arrested by this means, a brisk purge may be given, followed by guaiacum, if there is a history of rheumatism, and anodyne poultices applied externally. Gargling the throat with warm water and the inhalation of steam often afford great relief, and help to increase the rapidity of the suppurative process, the formation of an abscess, and its ultimate rupture and discharge of its contents. The after-treatment will consist in the administration of tonics, quinine, iron, etc.

By way of prophylaxis, all muffling up of the throat must be avoided, and the neck and chest sponged daily with cold water.

DISEASES OF THE ŒSOPHAGUS.

Affections of the œsophagus are generally due to attempts to swallow hot or corrosive fluids. The œsophagus may, however, be the seat of cancer, of inflammation not due to the action of irritants, and of stricture, the last in most cases being an after effect of inflammation. The œsophagus is also liable to stricture from spasm of the muscles, without any organic change in its walls. Whatever may be the cause or the nature of the disease, there is one well-marked symptom always present—dysphagia, or a difficulty in swallowing. The voice may also become husky. External pressure from aneurisms, enlarged glands, bronchocle, and new growths, may cause difficulty of swallowing. Care should, therefore, be taken in forming a diagnosis.

Treatment.—The treatment will depend upon the nature of the affection; if inflammatory, soothing bland drinks are indicated, together with nourishment in a fluid condition.

The treatment of the stricture will depend on its nature; if the product of inflammation, and lymph be deposited, an attempt may be made to promote absorption of the inflammatory products by daily passing a bougie down to and through the stricture, and using a bougie a size larger each time. Painting the throat with the tincture of iodine, or the inunction of the iodide of lead ointment may be tried. If due to spasm, tonics and anti-spasmodics will be required. In the case of cancer there is no treatment beyond relieving the patient's suffering.

Table showing the connection between certain Symptoms of Diseases of the Stomach and of other Organs.

Gastralgia. Cardialgia. Gastrodynia. Pain.	Pain is a marked symptom of diseases of the stomach, but gastric pain may be due to organic or functional disease of the colon, to aneurism of the abdominal aorta, and incipient disease of the vertebræ. Care in diagnosis is therefore required. Pain may also accompany emptiness of the stomach, relieved by the ingestion of a small quantity of food. Pain after taking food may be due to organic causes, ulcer, cancer, or functional in hysterical persons and those of weak digestions. For the diagnosis of ulcer and cancer, see the account of these diseases.
Alteration of Appetite. *Anorexia*–Loss of Appetite.	Loss of appetite may be due to loss of digestive power, or to organic disease. Loss of appetite is common in fevers and most morbid states not necessarily connected with the digestive organs.

Bulimia—Insatiable Hunger.

Bulimia (*βους* "an ox," and *λιμος* "hunger," or *βυ* and *λιμος*). Excessive hunger may be due to worms, diabetes, and disease of the mesenteric glands. It may also accompany one of the forms of gastric pain.

Pica—Depraved Appetite.

Depraved appetite for plaster and for dirt of all kinds is seen in some children, and in hysterical and pregnant women.

Regurgitation.

May occur in the forms of eructations or belching of gas or small quantities of food. Pyrosis or waterbrash is a form of eructation, accompanied with the discharge of a quantity of acid alkaline or neutral fluid from the stomach. The disease is more common in Scotland than in England. Some consider pyrosis as a result of a catarrhal affection of the mucous membrane of the stomach, accompanied with hypersecretion of gastric fluid; others, to excessive secretion of saliva, swallowed and arrested at the cardiac end of the œsophagus, and then regurgitated.

Vomiting. Emesis.

Common in some forms of dyspepsia preceded by nausea—(*ναυς* "a ship"—sea-sickness). Cancer and ulcer of the stomach. Pregnancy, and among hysterical women. Has been known in the *incipient* stage of *phthisis*, in *Addison's disease* of the suprarenal capsules, *Bright's disease of the kidney*, and in *cerebral diseases*. Care must therefore be taken in referring vomiting to its proper source.

Palpitation. Cough. Lingultus (Hiccough).

Palpitation of the heart, cough, and hiccough may all accompany gastric disorders. I have seen troublesome hiccough during an attack of gouty dyspepsia.

Hæmatemesis.

This may occur in certain *general diseases*—purpura, scurvy, cirrhosis of the liver, acute yellow atrophy of the liver, disease of the spleen, etc. It may occur as a form of vicarious menstruation. It is chiefly in simple ulcer, and in cancer of the stomach that hæmatemesis occurs. See the account of both these affections. It must be remembered that the stomach may contain a large quantity of blood, accompanied by all the signs of hæmorrhage, and yet hæmatemesis be not present. In *simple congestion* of the stomach, dependent in most cases on latent cirrhosis of the liver, hæmatemesis may occur.

DYSPEPSIA.

Lat. *Dyspepsia.* Fr. *Dyspepsie.* Ger. *Dyspepsie.*

Definition.—A term used to include those functional derangements of the digestive organs, depending either upon an unhealthy condition of the organs themselves, or upon the nature, quantity, and condition of the alimentary substances taken into the stomach.

Dyspepsia may be divided into Acute and Chronic.

Acute Dyspepsia:—Acute dyspepsia, called by the French *embarras gastrique*, and sometimes described as a *bilious attack*, arises from the ingestion of some very indigestible food, or from the sudden arrest of the functions of the stomach by strong mental emotions, or violent exercise after a full meal. There is a sense of weight and oppression at the epigastrium; nausea and perhaps vomiting may take place. When vomiting occurs naturally, the patient soon expresses himself relieved. Oppressive frontal headache is also present. The tongue is furred, a bitter taste is experienced in the mouth, and sooner or later diarrhœa occurs, accompanied with pain in the bowels. After the acute symptoms have subsided, there is generally some loss of appetite, and want of tone of the system generally.

Treatment.—The treatment consists in the administration of an emetic or a brisk purgative in the first stage, followed by tonics and medicines to relieve the gastric derangement and aid digestion.

Chronic Dyspepsia.—The symptoms of chronic dyspepsia are either local, that is, referable to the stomach or intestines; or general, that is, sympathetic or attendant. Digestion is of two kinds, *gastric* and *intestinal:* the former takes place in the stomach, and requires from two to four hours for its completion; the latter is a continuation of the former in the small intestines, where the food is acted upon by the bile, and by the pancreatic and intestinal secretions. The causes of indigestion are over-feeding, imperfect mastication, want of exercise, or excessive exertion indulged in too soon after a full meal; the inordinate use of tobacco or spirits, and all irregularities which tend to impair the digestive functions.

When indigestion is due to defective secretion, uneasiness, and ultimately acute pain, is complained of after meals, unaccompanied with any marked tendency to flatulence; the bowels are also more often relaxed than constipated. Should, however, the indigestion be the result of impaired motion, there is distressing flatulence accompanied with a sense of distention and uneasiness, but not of actual pain as in the former case, and constipation is the rule. In both cases there is loss of appetite, with a flabby pale tongue and unpleasant taste in the mouth. Sometimes an attack of eczema may occur during the course of a dyspeptic attack; improvement in the digestion will remove this complication.

For the sake of simplicity we may treat of chronic dyspepsia under five heads :—

1. GASTRODYNIA, a severe pain or cramp in the stomach and chest, coming on after taking any food, either of the smallest quantity or of the simplest kind. The bowels are constipated, and there is loss of appetite.

2. CARDIALGIA, or heart-burn, due to excessive formation of acid in the stomach, accompanied with a sensation of scalding extending into the throat.

3. SYMPATHETIC PALPITATION OF THE HEART, attended with much mental distress and dread of heart disease; all assertions to the contrary are of no avail, the patient believes that his heart is diseased, and from that belief there is no appeal.

4. PYROSIS, or water-brash, characterised by the frequent eructation of a clear fluid from the stomach into the mouth, sometimes acid, but at other times alkaline or neutral (*vide* Table).

5. DYSPEPSIA, *due to the presence of sarcinæ ventriculi* in the stomach, which gives rise to frequent nausea and vomiting of frothy sour-smelling fluid, in which the microscope detects the vegetable growth.

Dyspeptic patients try the patience of the medical attendant, for nothing seems to do any good; all his suggestions are frequently met with the statement that all have been tried before. In any of the forms of dyspepsia which occur in practice, the most distressing symptoms with which the physician has to deal are those to which the terms mental or nervous have been given.

Treatment.—The indications for the treatment of dyspepsia are threefold:—1. The dietetic. 2. The mental or moral. 3. The medicinal.

DIETETIC.—Many cases of dyspepsia may be cured by simple attention to the diet alone. No rule can be given that is applicable to all; the patient in most cases knows best what agrees or what does not agree with him. But all vices should be removed—inordinate smoking or drinking must be discontinued. The following diet may be suggested in some cases:—

Breakfast.—Cocoa and dry toast, and a lightly boiled egg.

Dinner.—A little meat, a little baked potato, and a tablespoonful of brandy in half a tumbler of water.

Tea.—As for breakfast.

Supper.—A little tripe or meat and bread, a tablespoonful and a-half of brandy in a tumbler of water.

The MENTAL or MORAL treatment of dyspepsia consists in establishing confidence in the patient, by a careful hearing and attention to the history of the case. Dyspeptics are generally "long-winded" in the enumeration of their symptoms, and are very tenacious of any inattention on the part of the physician. Change of air and scene may be tried.

MEDICINAL.—In gastrodynia the treatment will consist in the application of a mustard plaster to the pit of the stomach, and the administration of small doses of nitrate of silver, care being taken that the remedy is not continued long enough to cause a blue coloration of the skin. When cardialgia or heart-burn is a prominent symptom, the administration of alkalies is indicated, the best being the nitrate or trisnitrate of bismuth, or the bicarbonates of potash and soda. Dyspepsia with acid eructations is best treated by the administration of the dilute hydrochloric acid given about an hour before each meal. As an aid to diagnosis with regard to the choice for remedies, acid or alkaline, a slip of test paper may be placed in the mouth of the patient, and if the reaction betoken acidity, acids, with some one or other of the bitter infusions, may be tried. I have found that dyspepsia, accompanied with acid eructations, is always best treated with the mineral acids before meals, but in some cases a few doses of an alkaline mixture after meals before beginning the administration of acids does good. Nux vomica or strychnine is also a very useful drug in the treatment of dyspepsia. A dinner pill composed of aloes, capsicum, rhubarb, and nux vomica, is a most useful medicine, and should be taken one hour before the meal. Hydrocyanic acid will often give relief when there is much palpitation of the heart. The pyrosis will also be benefited by the treatment recommended for heart-burn. The nausea and sickness, so often a distressing symptom in dyspepsia, will be best treated with small doses of morphia, creasote, and hydrocyanic acid. When vomiting is due to the presence of the *sarcinæ ventriculi*, the stomach should first be emptied with the aid of the stomach-pump, and this treatment followed by the exhibition of the mineral acids in bitter infusions. The sulphites of potash and soda, as recommended by Sir William Jenner, have also been found useful.

GASTRITIS.

Lat. *Inflammatio ventriculi.* Fr. *Gastrite.*
Ger. *Magenentzündung.*

Definition.—Acute inflammation of the stomach.

Inflammation of the stomach, as an acute affection of that organ, is a very rare disease. When it does occur, it may result from exposure to cold or the indulgence in indigestible food, but it is generally due to the action of irritant poisons, swallowed accidentally or given with criminal intent. The disease has been known to follow the inhalation of the dust from arsenical wall papers. Should the disease, however, be met with, and have proved rapidly fatal, the mucous membrane of the stomach will be found inflamed, the redness more or less arborescent, in some places punctiform. If the fatal termination be delayed, besides the redness, the mucous membrane may be found more or less softened, and covered with mucus.

Symptoms.—The symptoms are intense burning pain at the epigastrium. During inspiration, the pressure of the diaphragm on the stomach increases the pain. The tongue is red, and looks as if deprived of its proper coating, and glazed. Nausea and vomiting are always present, the vomited matter being mucus tinged with bile. Thirst is sometimes present, and the desire for cold water is urgent, though in many cases it increases the vomiting. The appetite is lost. Should acute gastritis terminate fatally, the end is generally marked by the regurgitation rather than vomiting of thick grumous coffee-ground matter; by hiccough, great prostration, coldness of the surface, and feebleness of the pulse.

In connection with acute gastritis, Dr Fagge gives the history of two cases of acute gastric dilatation, or rather "acute paralytic distension" of the stomach. The distended stomach in one case contained no less than *seven pints* of fluid removed by the stomach pump, which in this case confirmed the diagnosis made by percussion and palpation. The presence of a splashing sound on manipulation is also an important sign. The symptoms of acute gastritis are so well marked that there can be little danger of an error in diagnosis. It was formerly, however, confounded with gastralgia. The paroxysmal character of the latter disease, the absence of pain on pressure, and the relief often felt after partaking of food, will help to distinguish the two diseases. Do not, however, confound this disease with typhoid fever in its early stages.

Treatment.—The indications for treatment are to subdue the inflammatory action, and to give the stomach a rest. Leeches may be applied to the pit of the stomach, or mustard and linseed-meal poultices to the same part. Purgatives, except in *the very earliest stages*, are contra-indicated. Blisters may even be useful in some cases. The vomiting may be relieved by small doses of morphia, or ice may be sucked by the patient. Milk, barley water, gum water, or ice water may be given. Injections of strong beef-tea, and alcohol when necessary, should be ordered. By this means rest will be obtained for the stomach, and the strength of the patient sustained.

CHRONIC GASTRITIS.

Chronic gastritis is by no means an unfrequent complaint. The symptoms which point to this condition of the stomach are chiefly those which accompany some of the forms of dyspepsia—constant tenderness over the pit of the stomach, loss of appetite, and loss of weight due to the mal-assimilation of food from imperfect digestion. The appetite is lost, and pricking pains are complained of in the stomach. Nausea and vomiting are among the most important signs of this disease. Among the causes of chronic gastritis, when not following an acute attack, are, the too frequent use of powerful stimulants, the absorption of arsenic into the system, and starvation.

Treatment.—The treatment is chiefly dietetic. Milk, lightly-boiled eggs, gum water, and other mucilaginous drinks, should be given. Blisters over the region of the stomach will be found of use. Bismuth, hyoscyamus, hydrocyanic acid, the bicarbonates of potash and soda, and gentian, are the remedies on which the most reliance is to be placed.

GASTRIC ULCER.

Lat. *Ulcus ventriculi.* Fr. *Ulcère de l'estomac.*
Ger. *Chronisches Geschwür.*

Definition.—Ulcer of the stomach, the ulcer being small and circumscribed.

Ulcer of the stomach is not an uncommon disease. Ulceration may occur during an attack of acute gastritis. Gastric ulcer, however, often occurs without any previous inflammatory action beyond that which surrounds the ulcer. The ulcers are more or less round or oval, and vary in size from that of a pin's head to that of a sixpenny piece or larger. If the ulcer perforates the walls of the stomach, it may give rise to fatal peritonitis. This dangerous complication is often obviated by the gluing together—by the process known as adhesive inflammation—of the stomach to the liver, pancreas, or to some other part at the seat of the ulcer, thus preventing the escape of the contents of the stomach into the peritoneal cavity. There is a peculiar form of gastric ulcer which, as it has a marked tendency to perforate the stomach, is known as the "perforating gastric ulcer."

It is thus best described:—"In a well-defined case there is, in the region of the pylorus, a circular orifice of from three to six lines in diameter, with a sharp peritoneal edge, as if a round piece of gastric parietes had been punched out. When viewed from within, the loss of substance on the internal membranes of the stomach, and especially of the mucous layer, appears more considerable, so that the edges of the hole seem bevelled off from within outwards. The pyloric half of the stomach is the seat of the ulcer; it is most frequently found in the middle zone of this portion; it is oftener seen at the posterior than at the anterior surface, almost always near to, and frequently at, the lesser curvature; and it occurs in extremely rare cases only at the fundus" *(Rokitansky).*

Symptoms.—Those of chronic dyspepsia. The most prominent diagnostic symptoms are—*pain* felt *immediately* after anything has been taken into the stomach, tenderness on pressure over the epigastrium, vomiting and discharge of blood from the stomach. This last sign may not be present for some time, and not until a vessel has been opened by the process of ulceration. The pain, which is of a burning character, differs from that which accompanies dyspepsia in being contemporaneous with the ingestion of food, and both Drs Brinton and Budd are inclined to localise the situation of the ulcer from the locality where the pain is felt. Thus pain in the pit of the stomach points to an ulcer on its anterior wall; pain in the back, to its posterior wall. The position of the patient is also of some diagnostic value, as he usually lies on the side opposite to that of the ulcer. Pain due to dyspepsia generally follows an hour or two after a meal. The vomited blood is dark and grumous, due to the action of the gastric juice. The bowels are usually confined, and the motions contain more or less dark blood mixed with them. The patient emaciates and presents a worn and haggard expression of countenance.

On the differential diagnosis of gastric ulcer, see "Cancer of the Stomach."

Etiology.—The exact cause or causes of ulcer of the stomach have not been clearly made out, but the following have been suggested:—

Corrosive action of the gastric juice on parts of the gastric mucous membrane where stasis of the blood has taken place, due to—

- (*a*) Simple catarrhal inflammation.
- (*b*) Venous hyperæmia when there is obstruction to the gastric or portal veins.
- (*c*) Thrombosis or embolism of a small artery.

The stomach, according to physiologists, is prevented from being digested by its own secretion, by the circulation of healthy or alkaline blood in its tissues. When this circulation from any cause is arrested in any portion of the stomach, the gastric juice acts on the part, which becomes necrotic, and an ulcer is formed. Females are more liable than males; the feeble and the emaciated than the robust. The ulcer may heal by cicatrisation, or it may prove fatal by rupture, escape of the contents of the stomach, followed by peritonitis, by hæmorrhage, and by inanition.

Treatment.—To arrest the vomiting, to relieve the pain, to check the hæmorrhage, and to assist in the cicatrisation of the ulcers, are the indications for treatment. The diet must be nutritious, easily digested, and of a bland, soothing character. Milk, and bread sopped in it, and light-boiled eggs, will form important articles of diet. If possible, the complete rest of the stomach should be ensured by the use of nutritive enemas. Ice must be given when the hæmorrhage threatens to be severe. As far as medicines are required, opium will be found invaluable in diminishing the peristaltic action of the stomach, and thus aiding the cicatrisation of the ulcers. Bismuth will be found of service, and will often arrest the vomiting. Nitrate of silver in a pill is often of great value.

DYSPEPSIA.	GASTRIC ULCER.
Pain felt some hours after ingestion of food. Vomiting rare. Hæmatemesis seldom present.	Pain felt immediately on the entry of food into the stomach. Vomiting as a rule present, and as a rule gives relief to the patient. Hæmatemesis not unfrequent.

CANCER OF THE STOMACH.

Carcinoma is not, fortunately, so common as the last-mentioned disease. The pyloric end of the stomach is the part most frequently affected, and scirrhus is more often met with than the other kinds of cancer. When the cardiac end of the stomach is attacked the disease, as a rule, begins at the lower end of the œsophagus.

Perforation, occlusion of the openings of the stomach, hæmorrhage from rupture of an artery, and dilatation or contraction of the stomach, are among the principal effects produced by cancer in this organ. Death occurs generally from inanition.

Symptoms.—Apart from the usual cachectic appearance of the patient, the following symptoms should be carefully observed:—Pain of a lancinating character, constant vomiting, often of coffee-ground looking matter, due to the presence of blood from some ruptured vessel in the cancer, loss of appetite, discharge of blood from the bowels, and the presence of a tumour in the epigastric region, will assist in forming a diagnosis. The course of cancer of the stomach is not always so well pronounced, and in the earlier stages of the disease a diagnosis is not always possible; and there are some diseases with which cancer of the stomach may be confounded, especially when no tumour can be detected in the abdomen. It may be mistaken for chronic gastric catarrh. If the patient be young, the presumption is against the presence of cancer. In chronic gastric catarrh, the pain is less severe, and improvement, as a rule, rapidly follows treatment. Gastric ulcer may be mistaken for cancer. In the former, pain comes on, as a rule, in paroxysms *immediately* food is taken, and is followed by vomiting; in cancer, on the other hand, vomiting does not always follow the immediate ingestion of food, for the food may remain some hours in the stomach before it is vomited; and it is to be noticed that vomiting relieves the pain in cases of cancer. And again, vomiting may disappear for a time, and only recur when some food difficult of digestion is arrested in its passage out of the stomach by the contracted pyloric orifice. Erosion of the gastric surface of the tumour may give rise to slight hæmorrhage from rupture of a capillary, and in these cases cancer cells may be detected in the vomited matters. In fat persons it may be almost impossible to detect the presence of tumour of the pylorus. When detected, its immobility points to its being a tumour in the walls of the stomach; but sometimes, when the tumour is very large, it may drag the pyloric end of the stomach below the umbilicus. The percussion note over a cancer of the stomach is usually dull, but tympanitic; a point of some value in diagnosis, which may help to distinguish it from cancer of the left lobe of the liver. In cancer of the stomach the food may be so long retained in the stomach before it is vomited as to become slightly decomposed. This is an important aid to diagnosis. The following complications may arise in scirrhus pylorus:—

1. Ulceration and hæmorrhage, suddenly ending fatally.
2. Jaundice from compression of the bile duct.
3. Ascites from pressure on the portal vein.
4. Hæmorrhoids, also arising from pressure on the portal vein.

In examining the abdomen, place the warm hand flat on the abdominal parietes and then make equable and gentle pressure. If you poke about with the fingers you will throw the abdominal muscles into contraction and obscure your diagnosis.

The **Treatment** is only palliative, but much may be done to prolong life for years by careful diet, and hydrocyanic acid to relieve the vomiting. Creasote is also useful an hour after food to prevent decomposition.

DILATATION OF THE STOMACH.

The stomach may become dilated as a result of cancerous disease of the pylorus, or of simple induration occurring in the walls of the organ at its pyloric end. But apart from these causes, dilatation of the stomach may occur from causes not yet clearly understood. I have already spoken of acute paralytic distension of the organ. Chronic dilatation is frequently accompanied by the presence of *Sarcinæ ventriculi*, and it has been attributed to hysteria, hypochondriasis, and over-eating. Careful palpation and percussion of the organ will assist in the diagnosis, together with the absence of symptoms peculiar to well-known diseases of the stomach. The treatment found most useful in gastric dilatation consists in emptying the stomach by the aid of the stomach pump, and then well washing out the organ with weak solutions of carbolic acid, permanganate of potassium, carbonate of soda, etc. This treatment has a wonderful effect in removing the vomiting, pain, and constipation from which the patient suffers.

ENTERITIS.

Lat. *Enteritis.* Fr. *Enterite.* Ger. *Enteritis.*

DEFINITION.—Enteritis is acute inflammation of the mucous membrane of the small intestines.

The inflammatory action in enteritis is generally limited in extent. The anatomical characteristics of this disease are those which belong to inflammation of other mucous membranes. The inflammation may vary in character from slight redness to the most intense crimson, ending in mortification.

Symptoms.—The symptoms of acute enteritis are abdominal pain, referred to the parts surrounding the umbilicus, tenderness on pressure over the abdomen, nausea, vomiting, and diarrhœa. Sometimes there is troublesome constipation. The patient usually lies on his back, with his knees drawn up to his stomach, in order to relax the muscles of the abdomen. In most cases we have the tenderness of peritonitis blended with the twisting pains of colic. The stools are watery, and often contain large quantities of mucus. The general symptoms are not well marked, but sometimes there is shivering and hot flushes and sweats; the pulse is quickened, the head aches, and there is sometimes much prostration. The tongue in most cases is normal; but as the inflammatory action proceeds, it may become coated, and ultimately dry and parchment-looking. The disease is usually a slight affection, very rare during adult life, but a common complaint with infants, in whom it may be complicated with thrush.

Enteritis must be distinguished from colic, peritonitis, dysentery, gastritis, typhoid fever, and hernia. The pain in colic is relieved by pressure; there is an absence of inflammatory symptoms, and constipation rather than diarrhœa is present in the early stages of colic. The diagnosis from peritonitis is more difficult; diarrhœa is less frequently a concomitant of peritonitis than it is of enteritis; rigidity of the abdominal muscles is absent in the latter, and the general symptoms are more aggravated in the former than in the latter: from dysentery by the absence of the characteristic dysenteric stools, of pain in the situation of the colon and tenesmus: from acute gastritis in this, that the pain in gastritis is referred directly to the region of the stomach: from typhoid fever, by the absence of the typical symptoms of that fever and the rose eruption.

To avoid mistaking enteritis for hernia, the usual seats of rupture should be examined. It should be borne in mind, however, that enteritis may end in mortification of the intestine; this condition is marked by the absence of pain, while the signs of depression continue, or become even more urgent. The pulse becomes intermittent, the countenance cadaverous, the skin bathed in a cold sweat; there is distressing hiccough, and the patient dies by slow asthenia.

Treatment.—The treatment consists in the administration of purgative enemata and the free use of opium. Hot fomentations or poultices over the abdomen will give great relief. Small doses of the hydrargyrum cum cretâ, with the compound ipecacuanha powder, are very useful in enteritis. The diet should be simple and of a bland, soothing nature, composed chiefly of milk. When necessary, stimulants must be given.

TYPHLITIS.

Lat. *Inflammatio cæci intestini.* Fr. *Typhlite.*
Ger. *Blinddarmentzündung.*

Definition.—Inflammation of the Cæcum.

Inflammation of the cæcum, known also as *typhlitis*, *typhlo-enteritis*, or *cæcitis*, may be due to an accumulation of fæces; to the lodging of extraneous substances in the colon or its appendix, which gives rise to inflammatory action, or to ulceration, as is most frequently the case, especially when the inflammation spreads from the surrounding tissues as it does in perityphlitis. The perforation may be due to typhoid, dysenteric, or tuberculous ulceration. As a result, an abscess may be formed in the iliac region, if the perforation takes place in that portion of the bowel not covered by the peritoneum. Peritonitis, of course, follows the perforation of the ulcer into the peritoneal cavity.

Symptoms.—Pain in the right iliac fossa, symptomatic fever, swelling in the region of the cæcum, and in severe cases, perforation of the intestine or abscess. On examining the abdomen the muscles will be found tense. This is a provision of nature to protect the inflamed tissues beneath. In perityphlitis, the resulting abscess may burst externally, giving rise to a fistulous opening, through which, if the bowel be involved, the contents of the bowel are discharged. Perforation of the *appendix vermiformis* may occur independently of cæcitis. More or less peritonitis or perityphlitis accompanies typhlitis, and it must be distinguished from ovaritis, psoas abscess following caries of the spine, renal abscess, and cold abscess occurring in the muscles of the abdominal walls.

Treatment.—The application of anodyne fomentations to the part affected, the administration of opium to relieve pain, and the use of enemata with castor oil, is the treatment to be adopted. Active cathartics are worse than useless; they are dangerous. The vomiting which often accompanies this affection is best treated by ice or effervescing drinks. In examining an inflamed cæcum be careful how you knead the part or you may rupture the bowel.

DIARRHŒA.

Lat. *Alvus soluta.* Fr. *Diarrhée.* Ger. *Durchfall.*
Syn. *Diarrhöe.*

Diarrhœa, or looseness of the bowels, is rather a consequence of certain pathological conditions than a disease in itself. It is characterised by the alvine evacuations being more frequent and more copious than in health.

An attack of diarrhœa may be due to indigestible food, to cold or damp, to great mental emotion, or to the exhalations of decaying animal or vegetable substances. A fertile source of this affection is the emanations from foul drains, or the use of bad drinking-water.

Symptoms.—The symptoms which attend an attack of diarrhœa are, griping pains in the abdomen and constant desire to go to stool, which is most frequently followed by a sensation of relief. Not unfrequently during the discharge, there is a disagreeable sinking sensation in the abdomen. The purging may be accompanied with vomiting. Faintness also is not uncommon. The skin is cool, the pulse feeble and irregular. It should be borne in mind that diarrhœa often occurs in wasting diseases, as in the *colliquative diarrhœa* of pulmonary tuberculosis, and also in some fevers, as typhoid, etc.

Treatment.—The treatment will depend greatly on the cause of the attack. If due to over-eating—*diarrhœa crapulosa* of the old writers,—a dose of tincture of rhubarb or the compound rhubarb powder, or of castor oil, will best meet the requirements of the case. Pain, when present, may be relieved by opium. Astringents, sulphuric acid, catechu, kino, and many others, are indicated when the evacuations continue frequent, and the weak condition of the patient demands the speedy arrest of the discharges.

COLIC.

Lat. *Colum.* Fr. *Colique.* Ger. *Kolik.*

Definition.—Painful contractions of the muscular walls of the intestines, relieved by pressure on the abdomen.

An attack of colic generally accompanies or follows a fit of indigestion. It is characterised by paroxysms of severe twisting pain in the abdomen, usually felt round the umbilicus. The pain is *relieved by pressure*, so that the patient is in most cases found lying on his belly, or pressing his hand on his abdomen. Not unfrequently the desire to go to stool is urgent, but nothing passes but wind. The patient may be troubled with eructations of gas smelling like burnt feathers. The pulse is unaffected unless the pain is very severe and has continued for some time, when it may become so from the anxiety of the patient. The cause of colic is frequently attributable to the local action of indigestible food, which gives rise to the painful spasm that is the characteristic symptom of this disease.

It differs from dysentery by the absence of the characteristic dysenteric stools; from enteritis and peritonitis by the absence of inflammatory symptoms, and by the fact that the pain is relieved by pressure.

Treatment.—The treatment usually adopted is to give an aperient and opiate at the same time. A dose of the tincture of rhubarb, together with chloric ether, aromatic spirits of ammonia and the tincture of opium, will often give almost immediate relief. If colic follows a full meal, an emetic may be given with advantage. Injections of gruel containing half a drachm to a drachm of the tincture of opium, and the same quantity of the tincture of assafœtida, may be given. Colic forms the only exception to the rule laid down by Dr Wilkes "that whenever a pain in the abdomen is so severe as to cause a patient to send for a medical man, this *ipso facto* proves the administration of a purgative to be unjustifiable."

LEAD COLIC.

Lat. *Colum ex plumbo.* Fr. *Colique de plomb.*
Ger. *Blei-Kolik.*

Definition.—A form of colic due to lead poisoning and characterised by constipation and a blue line along the margins of the gums close to the teeth. Dropped-hand or paralysis of the muscles of the forearm occurs in the chronic form of the disease.

Painters' colic *(colica pictonum)* is a disease due to lead poisoning. It is known in the West Indies as the "dry belly-ache."

Symptoms.—An attack of painters' colic comes on slowly; there is an anæmic condition of the blood, a fœtid odour of the breath, and metallic taste in the mouth. The appetite is considerably impaired or entirely lost, the bowels are constipated, and pain is felt in all the limbs. More or less emaciation and muscular debility are also present. Pain, like that which attends common colic, is felt in the abdomen, chiefly about the umbilicus; at first it is slight, but it soon becomes severe. There is always more or less flatulence. The presence of a blue line along the gums, close to the teeth, is a diagnostic symptom of some value. A peculiar paralytic affection of some of the voluntary muscles—principally those of the fore-arm—is often present, and is commonly known as "dropped-hand." The lead may find its way into the system through the lungs, skin, and stomach.

Treatment.—The treatment is palliative and curative. Anodynes to relieve the pain are indicated. The administration of sulphuric acid has been recommended by Gendrin, and its value confirmed by Dr Bennett. Sulphuric acid, diluted with water and sweetened with the syrup of lemons, has been used with considerable success as a prophylactic in lead manufactories. Sulphur baths are also useful to remove the lead from the skin. Lately, the galvanic bath has been used at St Mary's Hospital, with the most favourable results. The iodide of potassium in gradually increasing doses has been tried, and has met with many advocates for its use. Purgatives, especially the sulphate of magnesia, are sometimes necessary, and may help to remove the lead from the alimentary canal.

CONSTIPATION.

Lat. *Alvus adstricta.* Fr. *Constipation.* Ger. *Verstopfung.*

DEFINITION. — Functional affection of the large intestine, attended with insufficiency of the evacuations from the bowels. The term *Costiveness* has also been applied to this condition.

Constipation is incidental to many affections.

It may give rise to pains in the head, accompanied with a sensation of weight or pressure on the head. Pain, with distension of the bowels, is not unfrequent. Pain and straining accompanying the act of defecation are often present, and may become very distressing.

The presence of hæmorrhoids has been attributed to this condition of the bowels.

During health it would appear that one free daily evacuation is necessary; but this, though the rule, is not without many exceptions, and yet the health remains unimpaired.

The causes of costiveness are neglect or disregard to the requirements of nature; by this means the gut becomes abnormally distended, and its contractile power considerably impaired.

Treatment.—The treatment will be partly dietetic and partly medicinal. The dietetic will embrace the use of vegetables, bran bread, figs, prunes, tamarinds, etc. etc. Active exercise should be taken.

When medicines are used, aloes combined with some tonic, such as the extract of nux-vomica, or small doses of strychnine combined with the purgative, should be given. A valuable medicine will be found in the following pill:—℞. Ext. Aloes (Barb.), gr. iii; Ext. Nucis Vom., gr. ½; Mastiche, gr. ½. Mix. Fiat pil. Sumat unam nocte maneque. Preparations of iron are useful when added to a purgative. Aperient medicines should not be too long continued, and they should be dispensed with as soon as a habitual action of the bowels has been procured.

OBSTRUCTION OF THE BOWELS.

Obstruction of the bowels may be due to a variety of causes. The following Table, copied from Dr Tanner's *Practice of Medicine*, will assist in clearly understanding them:—

1. *Inter-mural*, or those originating in, and implicating, the mucous and muscular coats of the intestinal walls—
   (*a*) Cancerous stricture.
   (*b*) Non-cancerous stricture, comprising—1. Contraction of cicatrices, following ulcerations. 2. Contraction of walls of intestine from inflammation, non-cancerous deposit, and injury.
   (*c*) Intussusception.
   (*d*) Intussusception, associated with polypi.

2. *Extra-mural*, or those causes acting from without or affecting the serous covering—
   (*a*) Bands and adhesions from effusion or lymph.
   (*b*) Twists or displacements.
   (*c*) Diverticula.
   (*d*) External tumours or abscesses.
   (*e*) Mesocolic and mesenteric hernia.
   (*f*) Diaphragmatic hernia.
   (*g*) Omental hernia.
   (*h*) Obturator hernia.

3. *Intra-mural*, or obstructions produced by the lodgment of foreign substances—
   (*a*) Foreign bodies, hardened fæces, concretions having for their nuclei gall-stones, etc.

The following, according to Dr Brinton, are the usual seats of stricture of the large intestines:—Out of 100 cases, 4 occurred in cæcum, 10 in the ascending, 11 in the transverse, 14 in the descending colon, 30 in the sigmoid flexure, and 30 in the rectum.

Intussusception is most frequent in adults in the jejunum and ileum.

In children ileo-cæcal invagination is most common and generally occurs, before seven years of age.

Symptoms.—The symptoms of obstruction of the bowels are, pain more or less localised, and constipation. Vomiting occurs sooner or later, and is a prominent and persistent symptom. The vomited matter is at first mucous tinged with bile, but it may become stercoraceous or fæcal, if the obstruction be not soon removed. The pulse becomes quickened, the countenance anxious, and the body bathed in profuse perspiration. The complexion is muddy, and the urine is either suppressed or abundant, some relationship being noticed between the quantity of the flow and the seat of the obstruction; some ascribing it to the constriction being high up and preventing absorption of fluid, others to the repeated vomiting when the stricture is high up, and some to the influence of the sympathetic system. Blood may be passed with the stools. The abdomen is hard, enlarged, and tender to the touch, and more or less tympanitic. Should the obstruction yield to treatment, free and copious evacuations mark the return of the gut to its place, and the freedom of the patient from immediate danger.

The **Prognosis** is always unfavourable. Sometimes a cure may be affected by the sloughing away of the invaginated portion of the gut, and by its being passed by the rectum. The process by which it is detached gives rise to adhesive inflammation, thus gluing the separated edges together, and so preserving the continuity of the bowel. Peritonitis may occur from the imperfect union of the gut where the process of sloughing has taken place, thus allowing the escape of its contents into the peritoneal cavity.

Treatment.—The treatment should consist in the administration of opium to lessen the peristaltic action of the intestine and relieve spasm. Cathartics are at all times contra-indicated. In all doubtful cases be sure to examine the usual seats of hernia.

Nourishment in a concentrated form should be given, and the patient's strength carefully sustained. The inflation of the intestines has been recommended by Dr Tanner, when the invagination occurs below the ileo-cæcal valve.

INTESTINAL WORMS.

The intestinal worms are divided under two heads—the Tape-worms or Cestodes, and the Nematodes. The former class includes—(*a*) Tænia solium; (*b*) Tænia mediocanellata; and (*c*) Bothriocephalus latus. The latter includes—(*a*) Ascaris lumbricoides; (*b*) Ascarius vermicularis; and (*c*) Tricocephalus dispar.

Ascaris, also *oxyuris vermicularis*, or thread, pin, or seat worm, is very small, usually not more than a third or half-an-inch in length, inhabiting the large, but rarely entering the small, intestine. It exists in large numbers, and generally occurs in children, in whom it gives rise to intolerable itching about the anus.

Ascaris lumbricoides attains a length of from four to six inches or more. They are of a whitish or pale straw colour; their bodies are round, and taper towards each extremity. They are not unlike the common garden worm, but are finer in texture and not ringed. Children are more subject to them than adults, though they are by no means uncommon in the latter. The small intestines are their usual habitat; they, however, occasionally make their way into the stomach, and are then frequently vomited. They seldom exist singly, but are often present in considerable numbers.

Symptoms. — The symptoms which indicate their presence are, irregularity of the appetite, which is often voracious, an offensive breath, and a complexion of a muddy hue. The teeth are frequently ground during sleep, which is always more or less disturbed. The belly becomes swollen, tense, and hot; the stools are unhealthy, consisting chiefly of mucus. The attention may be first drawn to them by their presence alive in the evacuations.

Treatment.—The treatment adapted for the removal of the *oxyuris vermicularis* consists in the administration of a brisk purgative—calomel or scammony—followed by small doses of the tincture of steel, or some other tonic. Scammony is perhaps the best purgative, as it not only clears away the worms, but also the unhealthy mucus which surrounds them, and in which their ova is lodged. As they occur mostly in ill-fed and ill-nourished children, a nourishing diet is required, together with the proper measures to improve the general health of the patient. Injections of salt and water, turpentine, or infusion of quassia, are also very useful. For the *lumbricoid* worms three or four grains of santonin, combined with a purgative, will often remove them. Turpentine is also a valuable vermifuge when given by the mouth, and may be given in half-ounce doses to adults.

TÆNIA SOLIUM, TÆNIA MEDIOCANELLATA, AND BOTHRIOCEPHALUS LATUS.

Tænia.—The *tænia solium* was once considered as the ordinary tape-worm met with in this country, but Dr Spencer Cobbold thinks that the *tænia mediocanellata* is the more common of the two. The *bothriocephalus latus* is generally found in Holland, Russia, Poland, and Switzerland.

The natural history of the tape-worm is of considerable interest, for there can be no doubt that the *cysticercus cellulosæ*, or *pork measle*, is the ancestor of the *tænia solium*, as the *cysticercus* of the ox is that of the *tænia* *mediocanellata.* The *cysticercus cellulosæ*, the presence of which in the flesh of the pig gives rise to the disease known as pig measles, is a small bladder worm, the head of which may be protruded from the bladder. Measly pigs may be known by the swelling of the inner surfaces of the eyelids and the under surface of the tongue, and also by the yellow specks in the angles of the eyes, their thick necks and lean loins. If the pork containing these worms be eaten when imperfectly cooked, they become tape-worms in the stomach of the eater. The head of the *tænia solium* is about the size of a pin situated at one extremity of a thin neck, which broadens out into the tape-like body of the animal, and which is composed of oblong, flattened segments, each exactly resembling the other. Each segment contains both male and female sexual organs, the segment in time being thrown off to deposit its ova. The rounded head of *tænia solium* has four suctorial discs, and a double circle of hooks; that of *tænia mediocanellata* is without the hooks. The head of *bothriocephalus latus* is club-shaped, with no suckers, and only a longitudinal slit. The tape-worm may grow to many yards in length. The presence of the worm is generally known by the appearance of the cast-off segments in the stools, sometimes attended with considerable constitutional derangement.

Treatment.—A dose of castor oil should be given early in the morning, and repeated at night if necessary. During the day the patient should be limited to slops, no solid food being taken; on the following morning a full dose of the oil of male fern, mixed in a little honey or syrup, is given, and the castor oil repeated in the evening. The object of this line of treatment is to clear the intestine, and expose the worm to the full influence of the poison. Other anthelmintics used in the treatment of the tape-worm are—kusso, the dried flowers of the *brayera anthelmintica*, the bark of the root of the pomegranate, kamela (consisting in the hair from the capsules of Rottlera tinctoria), and many others.

TRICHINOSIS.

Lat. *Trichinosis.* Fr. *Trichinose.* Ger. *Trichinenkrankheit.*

DEFINITION.—Trichinosis, or trichinous disease, is an affection due to the presence of a small encysted *entozoon*, the *trichina spiralis* of Owen, imbedded in the muscles.

The encysted *trichina spiralis* is found embedded in the fibres of all the striped muscles of the trunk and limbs, and even in the heart, where it appears in the form of white ovid bodies or capsules, the capsules being sometimes calcareous. The worm passes the greater part of its existence in the chrysalis state in the muscular system of one animal, and only reaches its mature condition in the stomach of another. Virchow and Zenker assert that the trichina not only frequently presents itself in the human organism, but that this organism is most favourable for its full development. Once in the stomach, the period of incubation is about six or eight days, and then propagation rapidly begins and continues, so that Dr Kellen estimates that in a few days after the ingestion of half-a-pound of meat the stomach and intestines may contain thirty millions of the worms. The worms when introduced into the stomach leave their capsules, which are dissolved by the gastric juice, become free, and produce young. The young worms then leave the stomach through its coats for the muscles, where they become encysted. The trichina is most frequently found in pork, seldom in sheep, horses, or oxen, the last being the freest. The disease has made its appearance in several countries, but it was in Germany where it first attracted attention, and was there attributed to the use of pork in which the disease was present.

Symptoms.—Intestinal irritation, loss of appetite, sickness, malaise, general weakness of the limbs, and diarrhœa. The eyelids swell as well as the joints, the skin is bathed in a cold clammy sweat, and a low form of fever sets in. Death may be due to peritonitis, paralysis of the muscles, the result of their destruction, or to irritative fever. During the perforation of the coats of the stomach and bowels by the worms, the mucous membrane becomes inflamed, and pus is formed on the surface, and the stools become bloody. When the worms become encysted, the acute symptoms abate, and the patient recovers—recovery being due to the smallness in the number of the worms originally taken into the stomach. This disease must not be confounded with typhoid, acute rheumatism, or acute tuberculosis.

Treatment.—The treatment is more preventive than curative. The prevention of the disease consists in total abstinence from uncooked pork. Smoking, pickling, or salting cannot be relied on, as no temperature below 192° F. appears to be effectual in the destruction of the *trichina spiralis.* Professor Mosler, of Berlin, recommends the internal administration of benzine, suspended in syrup or mucilage, as a means of cure. Cathartics should be given in the first instance, in order to expel, if possible, the worms from the alimentary canal.

DISEASES OF THE ABDOMEN.

PERITONITIS.

Lat. *Peritonitis.* Fr. *Péritonite.* Ger. *Bauchfellentzündung.*

Definition.—Peritonitis is acute inflammation of the peritoneum, or serous membrane which lines the inner surface of the abdominal walls, and by which the intestines and organs of the abdomen are surrounded.

Peritonitis may arise from external injury, from the effects of cold, or the extension of diseased action from neighbouring structures, ulceration of the intestines in typhoid fever, or lastly, it may be the sequel to severe and complicated parturition.

Symptoms.—The commencement of the disease is often marked by successive chills or rigors, accompanied with intense pain, generally starting from one point and rapidly spreading over the entire abdomen, which becomes swollen, hot, and tense, and excessively tender to the slightest touch. The patient lies on his back with his knees drawn up, in order both to relax the abdominal muscles and support the weight of the bed-clothes. The countenance wears an anxious expression, the breathing is short, quick, and thoracic, being performed chiefly by the intercostal muscles. Nausea and vomiting are continually present, consisting of the ejection of bilious fluid. The bowels are confined, and the urine may be retained or suppressed. The pulse is small and very quick, sometimes as frequent as 120 beats a-minute, and the tongue is white and creamy. The temperature varies from 100° to 105° F. An important characteristic of peritonitis is the marked tendency to sudden collapse. Towards a fatal termination the tenderness of the abdomen may suddenly undergo great diminution, the extremities becoming cold, the face pinched and anxious, troublesome hiccough, quick pulse—140 to 160—and rapid collapse. The attack may run a rapid course, and may prove fatal by asthenia in two or three days, sometimes in twenty-four hours.

The morbid appearances found after death in a person dead with peritonitis are, preternatural vascularity of the bowels, especially the small intestines, which are also distended with gas and fluid. The peritoneum and the convolutions of the intestines are all glued together by the effusion of plastic lymph, which always occurs when serous membranes are the subjects of inflammation.

Treatment.—The treatment now usually adopted is to give opium in a large dose at first, and then to continue its action by repeated small doses, alone, or combined with some preparation of mercury, given at short intervals. Hot linseed-meal or bran poultices should be applied over the entire surface of the abdomen. Flannels wrung out in hot water, then sprinkled with turpentine and applied to the abdomen, will often afford intense relief. A saline diaphoretic draught may be given, and repeated every four hours. Aperients should be avoided, but the use of soothing enemata will often be of great service. The object in view is to give, if possible, the bowels complete rest.

CHRONIC PERITONITIS.

Lat. *Peritonitis longa.* Fr. *Péritonite chroinquc.*
Ger. *Chronische Peritonitis.*

Definition.—Chronic inflammation of the peritoneum.

Chronic peritonitis may be the sequel to the acute attack, but is more frequently a scrofulous disease, attended with a deposit of tubercles in and under the peritoneum. It generally occurs in young adults of cachectic appearance.

Symptoms.—The symptoms are somewhat obscure. Occasional pain and tenderness of the bowels are not unfrequent. The abdomen gradually enlarges, and acquires a peculiar irregular and knotted feel. The patient emaciates, and finally becomes hectic, and dies after perhaps some years of suffering. The morbid appearances which are found after death are—the presence of old adhesions, the bowels glued together, the liver covered by a thick false membrane, and tubercles scattered about through the abdomen.

Treatment.—The treatment must be regulated by the symptoms and condition of the patient. The exhibition of the iodide of iron or the iodide of potassium and cod-liver oil is advisable. Change of air often aids the other measures of treatment. Poultices or blisters to the abdomen will often be necessary. In one case of tubercular peritonitis I cured the patient with a blister, but she died two years after of obstruction of the bowel, due to a band of lymph. I believe that in this case an earlier application of the blister would have prevented the formation of lymph and the fatal result.

ASCITES.

Lat. *Ascites.* Fr. *Ascite.* Ger. *Ascitis.*
Syn. *Bauchwassersucht.*

DEFINITION.—An effusion of fluid into the peritoneum, whether the result of inflammation or not.

The terms *ascites*, *hydro-peritoneum*, or *peritoneal dropsy*, are also applied to this condition.

The presence of lymph is characteristic of an effusion due to inflammation; its absence, therefore, marks the true non-inflammatory origin of ascites. Dropsy is not a disease in itself, but is rather the effect or symptom of disease. Ascites is generally due to disease of the liver — cirrhosis, cancer, or obliteration of the portal vein—to disease of the heart, spleen, or kidneys, and extreme debility.

Symptoms.—As a rule, the accumulation of fluid takes place without pain or any local symptom. The upper part of the body may be more or less emaciated, or look more so from the enormous size of the abdomen. The abdomen is greatly distended, and frequently presents a marbled or mottled appearance, due to the enlarged veins, which ramify on the surface. Anasarca of the lower extremities is often present. The scrotum and penis are also frequently œdematous. The mechanical pressure of the fluid gives rise to a feeling of oppression, and in some cases there is inability to lie on the back. The functions of the abdominal organs are more or less impaired, and very frequently they suffer considerable displacement.

The urine, often small in quantity, contains bile when the dropsy is due to disease of the liver; albumen, when renal disease is present.

Care must be taken to avoid the error of mistaking ascites for an enlarged bladder; the introduction of a catheter will at once decide the nature of the case.

Pregnancy, and great corpulence, have also at times been mistaken for dropsy.

Ovarian dropsy must also be separated from ascites.

ASCITES.	OVARIAN DROPSY.
The abdomen is *equally* enlarged and fluctuating. In ascites the bowels float up through the fluid, in whatever position the patient may be placed. If the patient be lying on his back, a clear sound is elicited by percussion over the anterior surface of the abdomen. By changing the position of the patient and noticing the sound elicited on percussion, the diagnosis will be assisted.	The ovarian tumour is *more on one side than on the other*, and fluctuates less distinctly, being generally composed of one or more cysts. In ovarian dropsy the tumour occupies the anterior portion of the abdomen, the intestines being behind and on both sides of it. A dull sound is heard anteriorly on percussion when the patient is in a supine position.

Treatment.—The treatment will consist in the use of drastic purgatives and diuretics. In dropsy due to disease of the heart and the liver, purgatives and diuretics may be employed; but in renal dropsy, cathartics, combined with preparations of iron, should only be used. Digitalis, by increasing the blood pressure, acts as a valuable diuretic in cardiac dropsy. When these means fail, recourse must be had to *tapping.*

DISEASES OF THE LIVER.

The diseases of the organ now about to be described are, disorder of function, acute inflammation of the peritoneal covering, ending in adhesions; acute hepatitis of the structure, terminating in resolution or abscess; abscess, or purulent deposit due to pyæmia; cirrhosis, or hobnail liver; nutmeg liver; fatty degeneration of the liver; lardaceous or waxy liver; hydatid disease; malignant disease; and congestion, due to valvular disease of the heart, to hot climates, or intermittent fever.

Dr Murchison has suggested the following classification of the disease of the liver causing enlargement :—

Painless.—Fatty liver, hydatid tumour, hypertrophy, and amyloid liver.

Painful.—Congestion, catarrh of bile ducts, obstruction of common duct, with retention of bile, cancer, pyæmic and tropical abscesses. The painful enlargements are accompanied with jaundice, and are rapid in their course; in the painless enlargements, jaundice is absent, and the course is chronic. In all diseases of the liver do not neglect a careful inspection of the organ by palpation and percussion.

Anatomical Relations.—Situated in the right hypochondrium and filling up the hollow of the diaphragm upon this side, the liver is almost completely covered by the ribs, except the left lobe, which projects into the epigastrium. The upper margin under the nipple is about the fifth intercostal space, below the axilla, about the seventh intercostal space, and in the tenth intercostal space close to the spine. The position of the lower margin is not constant, for it may vary from being covered by the ribs to nearly three inches below them, without betokening disease. In percussing the liver adopt the following rules:—1. Do not percuss the liver when the stomach and intestines are loaded with food, as after a meal. 2. Remove the effects of obstinate constipation by a purge. 3. Remove accumulation of gas in the intestines. 4. Use a pleximeter and begin to percuss from the third intercostal space downward. 5. To define the lower margin percuss very gently with two or three fingers. 6. Do not be misled by the dull sound caused by a tense abdominal wall.

FUNCTIONAL DISORDER OF THE LIVER.

A short notice of one of the most common among the complaints which come under the notice of the physician will not be out of place here. Biliousness, hepatic dyspepsia or *lithæmia*, appears to be more or less connected with some functional disorder of the liver, for which it is not easy to clearly define the cause. At one time held to be due to a "torpid state" of the liver; at another to congestion of the portal system; and lastly, supposed by Murchison to be due not entirely to defective secretion of bile, "but also to an interference with the normal processes by which albumen is disintegrated in the liver. The result is that, instead of urea, uric acid, a less perfectly oxidised body, is formed." The symptoms are those well known to accompany an attack of "biliousness" or "of the bile." Dyspepsia, a sense of weight or discomfort in the region of the liver, pain in the right shoulder, bilious vomiting, "coppery" taste in the mouth, frontal headache, and a considerable discharge of lithates in the urine, constituting the affection known as lithæmia. The treatment consists in the administration of a few grains of blue pill, followed by a saline purge. Free exercise in the open air, and chiefly on horseback, if that form of exercise can be indulged in. The waters of Püllna, Friedrichshall, Carlsbad, are also of great use in the treatment of functional disorders of the liver. From the presence of large quantities of lithates in the urine, patients often become possessed with the idea that their kidneys are diseased. A few doses (grs. 15-20) of the citrate of potash will clean their urine and remove their fears.

ACUTE HEPATITIS.

Lat. *Hepatitis.* Fr. *Hépatite.* Ger. *Leberentzündung.*

DEFINITION.—Acute inflammation of the liver.

For the sake of convenience, under the above head will be considered inflammation of the peritoneal covering, perihepatitis, or of the gland itself.

Symptoms.—Acute hepatitis is in most cases ushered in by well-marked rigors or chills. Fever follows, accompanied with more or less pain in the region of the liver. On practising palpation over the right hypochondrium, a fulness is perceived, due to enlargement of the gland. By gentle percussion, the size and position of the liver may be made out. When the inflammation is limited to the peritoneal covering, the pain is of a lacinating or stabbing character; but when the glandular portion of the organ is affected, the pain is dull and deep seated. Attempts to move in bed increase the pain, and the patient cannot lie on the left side, preferring to remain on his back. Pain shooting into the right shoulder may or may not be present. The appetite is lost, the tongue is coated with a yellowish white fur, and nausea and vomiting are present in the majority of cases, causing the patient intense pain by the constant attempts to relieve the stomach. Jaundice may be present, but is not a constant symptom, a yellowish tinge of the conjunctiva being in many cases the only evidence of its existence. The urine is often albuminous, and when jaundice is present, contains bile. Headache is in most cases a distressing symptom. Drowsiness and delirium generally usher in a fatal termination.

Morbid Anatomy.—As in other cases where inflammation attacks a glandular organ, engorgement and exudation are the primary effects: the latter, if resolution take place, is absorbed; if not, an abscess is in most cases the result.

Causes.—Inflammation of the liver most frequently occurs in hot climates, and seldom attacks persons under the age of twenty. It may be due to the abuse of alcohol, to exposure to cold, or it may follow or accompany other diseases, as dysentery, pneumonia, etc.

Diagnosis and Prognosis.—Hepatitis must be distinguished from pleurisy, pneumonia, and intercostal neuralgia, by a careful attention to the symptoms. Acute inflammation of the liver is in most cases attended with considerable danger, especially if an abscess be formed.

Treatment.—This will depend upon the condition of the patient, and the severity of the inflammatory action. Bleeding, general or local, may be called for. The application of hot poultices of mustard or linseed, or even of blisters, will be found useful. Opium to relieve pain is often necessary. For the rest, the physician must be guided by general principles.

ABSCESS OF THE LIVER.

Lat. *Abscessus.* Fr. *Abcès.* Ger. *Abscess.*

Abscess of the liver, due to pyæmia, differs somewhat from an abscess the result of inflammation of the parenchyma of the organ. In the former case, two or more abscesses of small size are found, whereas in the latter case the abscess is large, sometimes containing a considerable quantity of pus. This may be true of the cases seen in England of pyæmic abscess, and there is good reason to believe that "the differences between the tropical and pyæmic abscesses may depend upon their different ages;" that is, that the cases of pyæmic abscesses seen here are generally attended with such terrible complications that life is cut short before they can coalesce and form the larger abscesses so well known in tropical countries.

The clinical history of pyæmic hepatic abscess is very obscure, nor can the diagnosis be made with any certainty. By some it is supposed that the frequency of abscess of the liver in India is due to the abscess being a sequel of dysentery, and consequent on the ulceration of the large intestine inflaming, or even opening, the mesenteric veins which form the roots of the portal vein. Be this as it may, it is worthy of note that most of the cases of hepatic abscess seen in this country are from India, and with a history of previous attacks of dysentery. Again, dysentery may and does occur in Europe without the formation of abscesses.

Pathology.—The cause of hepatic abscess is best expressed in the one word, "embolic." A portion of a clot from the ulcerated intestine is set free, and is carried to the portal vein, in one of the ramifications of which it lodges. The portion of the gland beyond the clot is deprived of blood, but not wholly so, for the part is to a certain extent supplied by the surrounding capillaries; but wanting the necessary *vis a tergo*, local congestion is the result. Due probably to the diseased condition of the clot, and to the passive congestion of the part, softening and disintegration of the tissues follow, and an abscess is the result. The abscess may become encysted by the formation of a capsule or "pyogenic membrane," and this is most likely to occur when the abscess is of long standing. That the unhealthy nature of the clot has much to do with the formation of the abscess, may be inferred from the fact that the plugging of a vessel does not of necessity lead to an abscess, unless the clot come from some unhealthy part.

Hepatic abscess may open externally through the abdominal walls, if the liver become glued by adhesions to the inner surface of the muscles. Sometimes the abscess may dry up, its contents becoming pasty or putty-like. More frequently, however, the abscess opens into the duodenum, stomach, or colon, but seldom directly into the peritoneal cavity, because serous membranes are prone to form adhesions, and the liver thus becomes connected to the nearest intestine by organised lymph; and as the mucous membrane lining the hollow viscera is disposed to ulceration, a communication is formed, and the contents of the abscess are discharged into the bowel. The liver may also become adherent to the under surface of the diaphragm, the upper surface of this to the lung, and so the abscess progresses in that direction until it opens into a bronchial tube, when the contents are expectorated by the patient, mixed with bile. These terminations in the cases of pyæmic abscess are rare, as death in most cases takes place before the abscess becomes cysted. In the large abscesses so common in Indian patients visiting this country, the abscess has been known to remain quiet for some time and then suddenly to assume its former activity. An hydatid cyst may suppurate, and an abscess be the result. Suppurative changes may also follow the course of the portal vein.

Symptoms.—If in the course of an attack of acute hepatitis the patient be suddenly seized with severe rigors, followed by hectic or suppurative fever, the formation of an abscess may be inferred. But an abscess may form in the liver unattended by any marked disturbance, till perhaps the patient vomits pus or passes it *per anum.* The symptoms will vary greatly with the position of the abscess; thus, if pointing on the under concave surface of the gland, persistent and uncontrollable vomiting may alone be present, due to pressure on the stomach. If, again, the abscess is deep-seated, pain may be entirely absent. Pain as a symptom is most marked when the capsule is the subject of inflammatory action.

Dr Maclean records a case (*British Medical Journal*, vol. ii., 1874,) of a lady who, after a chill, had slight hepatic symptoms—so slight as to cause little alarm. The liver was but little enlarged; there was hardly any pain, only slight fever. The stomach was irritable from the first. After a marked rigor vomiting became incessant, and the exhaustion of the patient so extreme that death appeared imminent. Suddenly about a pint of pus was vomited, the irritability of the stomach subsided, and the lady made a good recovery. In these cases the temperature may vary from 100° F. to 104·6° F. Thus, if we get a case having the following history and symptoms, hepatic abscess may with some probability be diagnosed:—History of a previous attack of dysentery, progressive emaciation, persistent high temperature, enlargement of the liver, constant vomiting, and slight, if any, pain. Vomiting may be absent if the abscess be small or on the convex surface of the organ. On the whole, then, the formation of hepatic abscess is most insidious, and the diagnosis at all times difficult.

The **Prognosis** is unfavourable, even in the most favourable cases.

Treatment.—The treatment will consist in supporting the strength of the patient, and by opening the abscess should it point through the abdominal walls, and a reasonable time be allowed for the formation of adhesions between the muscles and the liver, in order, if possible, to prevent the discharge of pus into the peritoneal cavity.

In all cases it is advisable to make at first a puncture with a small trochar or a fine pneumatic aspirator needle. Sir J. Fayrer mentions a case where an accidental blow by a friend in the region of the liver ruptured an hepatic abscess in a young man, followed by recovery.

CIRRHOSIS OF THE LIVER.

Lat. *Cirrhosis.* Fr. *Cirrhose.* Ger. *Cirrhosis.*

Definition.—Chronic interstitial inflammation, attended with an increase of the interlobular connecting tissue of the substance of the liver, and with ultimate diminution in the size of the organ.

The terms *cirrhosis, hobnailed* or *gin-drinker's liver,* have been given to this disease. In the first stage of the disease the size of the liver is not much affected, but as the disease progresses it becomes smaller, with a marked diminution in the weight, and with increased firmness of the gland, so that it feels hard and firm. The capsule becomes more or less opaque, of a greyish-white colour, with flattened projections varying in size from that of a pinhead to that of a pea. These projections are due to contraction of the abnormal fibrous tissue forcing the parenchyma into a number of nodules. When the nodules are small, the liver is said to be "granular." When cut into, a like appearance is observed in the interior. The decrease in size, and atrophy of the organ, is due to the absence of fat, and to the pressure excited by an abnormal amount of fibrous tissue between the hepatic lobes, which also, compressing the portal vein and its terminal branches, gives rise to congestion of the portal system, and dropsy. The gall ducts become compressed, giving rise to biliary congestion and a yellowish-white, dark-green, brownish, or bright yellow coloration of the organ. On section, none of the healthy appearance of the organ is seen.

By some, cirrhosis of the liver is regarded as a diffused, sub-acute inflammation of the areolar or connective tissue existing between the lobules, forming an extension of Glisson's capsule. By others, the change is considered to take place first in the hepatic cells. In accordance with this latter doctrine, the change in the structure of the liver is not due to inflammation, but to a process of degeneration having its origin in the secreting cells of the liver. This degeneration of the secreting cells as a primary cause of cirrhosis is denied by Wilks and Moxon.

"Hitherto we have spoken of this substance as fibrous tissue. Microscopic examination, however, shows in it elements responding to its active and encroaching nature; it is crowded more or less with small cells. It is often full of networks of biliary ducts, which have led to the supposition that in it an effort arises to restore the hepatic tissue again by the same outgrowth from the ducts as you know originally produced the hepatic tissue; further, it is loaded with large vessels easily injected from the hepatic artery, for the compression exerted by the new formation tells more upon the feebler current in the portal vein, so that the hepatic artery enlarges to compensate."

The secreting cells do not appear to be transformed into the fibrous tissue, nor do they become loaded with fat as in true fatty liver, but they undergo a true fatty degeneration. The capsule is not, as a rule, much thickened, but it will not unfrequently be found more or less coated by a thick layer of adventitious tissue, in m ostcases the result of spirit-drinking, and which tissue may be easily separated, leaving the true capsule adherent. This coat, the result of disease, powerfully compresses the liver, aiding materially in the diminution of its size. Here and there gaps will be found in its continuity, the result of the contracting efforts of the membrane. It has been considered possible by some that cirrhosis may result from simple peritoneal inflammation, especially in those cases occurring in the young, where there is no history of intemperance. May not these cases have a history of syphilis?

The acute peritonitis, sometimes following the operation of *paracentesis abdominis*, is probably due to the chronic form becoming acute. Disease of the heart, spleen, and kidneys often co-exists with cirrhosis.

Symptoms.—Till the appearance of ascites, there is in many cases little or nothing to direct the attention to the condition of the liver; the symptoms that are present are referable to the habit of spirit-drinking, which, in the great majority of cases, is the cause of this disease.

In habitual dram-drinkers the symptoms are those of functional derangement of the digestive organs. A furred tongue, a feeling of weight and discomfort at the epigastrium and in the region of the liver, loss of appetite, flatulence, heart-burn, irregularity in the action of the bowels, at one time confined, at another troublesome diarrhœa. The action of the heart is irregular, and the patient complains of palpitation and occasional faintness. Frontal headache, noises in the head, restlessness at night, the rest being broken by bad dreams. A troublesome mucus collects in the back part of the throat, and causes considerable annoyance to the patient, who is constantly trying to relieve himself.

As the disease progresses, the liver is found smaller and the spleen larger than usual. Hæmorrhage from the bowels may occur during the course of the disease, and may be the first symptom to draw special attention to the liver. At last dropsy sets in; the abdominal veins increase in size, and give a marbled appearance to the belly; and the patient dies from exhaustion or blood-poisoning.

Treatment.—Very little can be done by way of treatment beyond attempts to relieve the sufferings of the patient. Aperients should be given when necessary, and tonics in the hope of improving the condition of the digestive organs. All alcoholic stimulants should be avoided. A visit to Carlsbad or Marienbad may be tried. Small blisters applied over the liver are sometimes useful, and can be better borne than cupping or leeching. The dropsy may be removed by tapping, if the respiratory functions are very much impeded.

Table showing points of difference between Cirrhosis and Syphilitic Cirrhosis.

CIRRHOSIS.	SYPHILITIC CIRRHOSIS.
Chronic diffuse inflammation of Glisson's capsule and of the fibrous stroma of the liver, with hypertrophy and subsequent shrinking of the areolar tissue within and covering the structure of the gland. The liver becomes firm and then contracted, the capsule being so puckered in as to give a "hobnailed" appearance. The change is said by some to take place first in the hepatic cells, by others in the areolar connective tissue existing between the lobules, and forming an extension of Glisson's capsule. The colour of the liver when thus affected is that of impure bees' wax. The affection is known as *Gin-drinker's liver.*	Syphilitic Cirrhosis is generally a simple local induration of portions of the gland, the rest of the organ remaining healthy and hypertrophied, and without the formation of the characteristic nodules of cirrhosis. The chief characteristics of this disease are its strict localisation, the portions affected only undergoing changes. *Hepatitis gummosa* consists in the presence of whitish depressions, like cicatrices, extending into the gland, and filled with opaque yellowish nodules more or less rounded, and varying in size from that of a linseed to a walnut. To this form of the disease the term "botyroid," "grape-like," has been given. The nodules—"nodes"—are sometimes caseous, but the caseous matter may disappear, leaving nothing but fibrous tissue, or the consistence of nodule may be that of gristle. There is generally the history of tertiary syphilis.

FATTY LIVER.

Lat. *Jecur adiposum.* Fr. *Foie gras.* Ger. *Fettleber.*

Definition.—Fatty degeneration of the liver.

The liver during health contains more or less fat—two to three per cent.; it is therefore somewhat difficult to decide what may or may not be considered a healthy condition of the organ. In most cases of fatty liver, the liver is increased in size, and of a softer consistence than it is in health. The secreting cells are found full of oil, and the specific gravity of the organ is considerably diminished. Wilks and Moxon mention a case in which the liver floated, owing to the fat it contained. When handled it imparts an unctuous sensation to the fingers; and the fat may be extracted by burning a piece in the flame of a lamp, or boiling a piece in a test-tube, when on cooling the fat will rise to the surface of the liquid. By the aid of the microscope, the size of the fat cells may be determined in the portions of the gland affected. The deposit of fat takes place first in the outer zone of the lobules, and extends towards the centre. This disease is more common in females than in males; and in many cases it accompanies other diseases, as phthisis, pneumonia, and the continued and eruptive fevers, gastric, ulcer, and dysentery. A condition of the liver known as *nutmeg* or *myristicated* liver, in which the organ, when cut into, presents the appearance of a nutmeg, is of no clinical importance. Fatty liver generally occurs in persons fond of the pleasures of the table, and who take little or no exercise. It is also found in cases of poisoning by phosphorus.

Treatment.—The treatment will consist in adopting measures to strengthen the system; and in cases of over-feeding, to restrict the diet. Regulated exercise is also necessary.

ALBUMINOID, WAXY, OR LARDACEOUS LIVER.

Lat. *Jecur lardaceum.* Fr. *Foie lardacé.* Ger. *Speckleber.*

Definition.—The deposit of a morbid product having many of the characteristics of starch, or of albumen more or less modified.

This condition, which is not peculiar to the liver, but which may occur in other organs, has received various names—viz., *albuminoid*, *amyloid*, *waxy*, or *lardaceous* degeneration. Unlike scrofulus and tuberculous products, it is not a growth of new cells, but a deposit within the existing tissue elements.

The morbid material characteristic of this affection, about the nature of which opinions vary, is by some considered to be analogous to starch, as it gives a blue colour with iodide of potash and sulphuric acid; by others it is supposed to be albumen more or less modified. No true chemical analysis has yet been made of this substance, for it is found almost impossible to separate it from the nitrogenous investing membranes.

When the liver becomes affected it is greatly enlarged —eight to fourteen pounds—but still preserves its shape, if fat be not also present in abnormal quantities. It, however, tends to mould itself on other organs, as the kidney, and in some cases even on the spleen. A thin slice held up to the light is translucent, and looks as if soaked in glycerine. The organ is firm and resisting, the interior being dry and pale. The deposit is supposed by Frerichs and Virchow to take place within the hepatic cells; by Budd, to be infiltrated exterior to the cells; in the muscular fibre cells of the middle coat of the small arteries, and in the walls of the capillaries, thence extending into the secreting cells of the liver *(Wilks)*. Under the microscope the hepatic cells are seen much enlarged, irregular in form, their nuclei imperceptible, and many of them fused together in masses, together with a certain amount of fatty infiltration. The deposit appears to select the middle zone of the lobule at the entrance of the hepatic artery; and in this apparent choice of position it differs from fatty and pigmentary deposits — the former occupying the outer zone, the latter the centre, the lardaceous, as we have just said, the middle. The portal circulation is not affected; thus ascites does not accompany this condition of the liver. Dr Dickinson is of opinion that, as most of those who suffer from lardaceous disease of the viscera are the subjects of long-standing purulent discharges, so this deposit of lardaceous material may be the result of the fibrine being dealkalised by the large quantity of alkali passing off with the pus—pus being alkaline. The dealkalised fibrine is then deposited as lardaceous matter. This theory, though ingenious, breaks down in the case of those who have not suffered from the purulent discharges, and it fails to explain the reason why the lardaceous change is practically limited to the liver, kidneys, and lymphatic glands.

The disease occurs most frequently in young persons, especially in those who have a tendency to scrofula, or who have been attacked by syphilis, or suffered from chronic phthisis. Males are more often affected than females.

During the course of the disease, general dropsy is not unfrequent, and is often attended with uræmic symptoms.

The **Prognosis** is unfavourable, as very little can be done by way of treatment. A combination of the iodide of potassium and the iodide of iron has been recommended by Frerichs. The diet should be nourishing and the patient should be warmly clad. The dropsy, when present, must be treated on general principles.

CANCER OF THE LIVER.

Lat. *Carcinoma.* Fr. *Cancer.* Ger. *Krebs.*

Like other parts of the body, the liver is liable to become the seat of cancer; in fact, this organ ranks next to the uterus in its liability to this disease. The variety of cancer most frequently found is the *encephaloid* or *medullary;* and next in rotation, *scirrhus* and *colloid*, the last being extremely rare. Cancer of the liver frequently has a character intermediate between encephaloid and scirrhus, generally occurs as secondary to deposits in other parts, and is seldom seen as a single tumour, being far more frequently scattered about the organ in numberless round tumours varying greatly in size. The growth of the disease is in most cases very rapid, death taking place in a few months. Cancer in most cases makes its appearance on the surface of the gland, and if not seen there, its absence may be safely inferred. To the naked eye the lobules of the affected liver, instead of being of their natural tint, appear whitish or black according to the variety of the disease present; and their consistence is also altered. The nodules on the surface vary greatly in size; are cupped, due to degeneration, *necrobiosis* going on in the central portions, which may become so soft as to have the appearance of abscesses, and into which a blood-vessel may open, a blood cyst being thus formed. The central portion of the cancer mass may be deprived of blood by a cancerous plug being formed in the supplying blood-vessels, or as the margins are the parts most rapidly growing, they rise up around the centre, which thus becomes apparently depressed. According to Wilks and Moxon, the cancer grows into a large branch of the portal or hepatic veins, roughens the channel of the vessel which arrests the flow of the blood, and a clot is formed. Into this clot the cancer then grows, "and so a large, soft, carcinomatous plug is found filling the vessel. Such great soft cancerous clots have been found without any implication of the vessel's walls." As to the origin of cancer in the liver, "from what we have seen, we have no doubt that, as a rule, the cancer cells are developed by direct transformation of the hepatic cells." Cancer is not the addition of new tissue, but the transformation of already existing tissue, and has been stated by some to be largely due to the agency of the epithelial cells lining the small blood-vessels. Colloid, as a primary disease, is said never to attack the liver. The portal veins appear to be more frequently the seat of cancer than the hepatic veins.

Symptoms.—To the usual cachectic condition of the patient are added pain in the region of the liver, of a shooting, stabbing nature, enlargement and irregularity in the form of the organ, which may feel nodular on examination, œdema of the lower extremities, and progressive emaciation and loss of weight. Peritonitis localised over the liver, and due to the nodular masses of cancer, is often present. Jaundice is more frequently present than dropsy, and is usually persistent.

The treatment is simply palliative.

ACUTE ATROPHY OF THE LIVER.

Lat. *Atrophia acuta.* Fr. *Atrophie aiguë.*
Ger. *Acute atrophie.*

Definition.—A rapid and marked reduction in the size of the liver, accompanied with almost total destruction of the gland.

The names *acute* or *yellow atrophy of the liver*, and *malignant* or *fatal jaundice*, have been given to this disease.

The pathology of this disease is very obscure. It was considered by Rokitansky to be due to an excessive secretion of bile, which causes liquefaction of the tissue of the gland. By others, among whom may be mentioned Frerichs and Bright, it is believed to be due to parenchymatous inflammation. The latter opinion is probably the correct one.

Red atrophy is a chronic form of yellow atrophy, "the yellow being an acute or excessive intensity of the red" *(Wilks and Moxon)*.

Whatever may be the views entertained regarding the nature of this disease, it is a malady of great importance, and fortunately is of rare occurrence. The disease consists in the rapid and total destruction of the hepatic cells in every part of the gland, without the formation of new material. The chemical constitution of the liver is, however, changed, and large quantities of tyrosine and leucine are found.

Etiology.—The cause of the disease is not clear. Females—especially during pregnancy—suffer much more frequently than males. Syphilis and mental distress are said to be among the causes. It is so rare in children that Niemeyer says that "acute yellow atrophy never occurs in children." The disease has been known to occur amongst various members of the same family.

Symptoms.—The symptoms are—at first, simple derangement of the functions of digestion; followed by jaundice, intense pain in the head, active delirium, and convulsions; gradually a condition of stupor succeeds, ending in fatal coma. The head symptoms have been attributed by Virchow to uræmic poisoning, but it would appear that other causes are at work. The bowels are confined, the stools being firm, dry, clay-like, and deficient in bile, but towards the fatal termination, tarry from the presence of blood. The urine contains bile, and is sometimes albuminous. The temperature is not augmented, and in most cases is below the normal standard. The pulse varies in frequency, is at first slow, but on the outbreak of the nervous symptoms it increases in frequency to 110–120. Crystals of leucine and tyrosine are present in the urine.

The liver rapidly decreases in size, as discovered by palpation. The liver, which in health weighs between four and five pounds, may in this disease be reduced to a pound and a few ounces. The spleen, on the other hand, is congested and enlarged. The gall bladder is, as a rule, empty. Petechiæ and ecchymoses, due to extravasation, are often present.

The duration of the disease, from the commencement to a fatal termination, is generally one week, though life may be prolonged to four weeks. Most cases end fatally.

Diagnosis.—From typhoid, by the early symptoms of that disease, the roseolar eruption, bronchial catarrh, diarrhœa, and wandering delirium. Do not mistake meningitis, pneumonia, and peritonitis, accompanied with jaundice, for acute yellow atrophy. Bilious fevers are, as a rule, of a remittent type. Pyæmia is distinguished by rigors and purulent deposits. Phosphorous poisoning, by the history of the case, the aliaceous odour of the breath, and the luminosity of the vomited matters in the dark.

Treatment.—So little is known of the nature and cause of this disease, that not much can be said as to treatment. Drastic purgatives have been given, but with very limited success. When the vomiting is severe, measures to arrest it may be adopted; and symptoms as they arise must be treated as occasion requires.

HYDATID TUMOURS OF THE LIVER.

The liver is the most frequent seat of hydatid tumours. These tumours consist of a bladder contained in a sac to which it is non-adherent, so that the hydatid drops out of the containing cavity. The bladder contains a clear fluid, slightly saltish (sp. gr. 1010), in which float a number of round or ovoid cysts. These cysts may become almost bony, from the deposit of earthy matter in their walls. The relation of hydatids to tape-worm has of late been made out, and it would appear that the ova of the tape-worm are taken up by branches of the portal vein from the intestinal canal, and then carried into the liver, where they become developed into cystic animals. In most cases a number of smaller animals are contained in the larger. When the inner surface of the cyst is examined by the microscope, it will be found in most cases to have attached to it minute bodies looking like white grains. These are clusters of very minute round bodies enclosed in a very thin membrane. These minute bodies are found to be the heads of small tape-worms—*Tœnia echinococcus*—armed with hooklets and suckers. One half of the creature is capable of being tucked into the other half. It does not appear that these heads develop into mature tape-worms, but follow the law which has been found to hold good in the development of the tape-worm; that is, they require to be transplanted, so to speak, into another body before they arrive at maturity. The Tænia echinococcus is usually found in the dog. An hydatid tumour may become so large by absorption of the liver tissue as to push the diaphragm almost to the first rib. From some obscure cause or other these tumours may dry up, and the destruction of the hydatids and echinococci takes place; sometimes they burst, discharging their contents either into the thorax or into the alimentary canal.

Symptoms.—As a rule, the prominent symptoms will depend upon the direction the tumour takes. An hydatid tumour is often unsuspected during life, and its existence only revealed by a *post-mortem*. Peritonitis may be the result of their presence, or the hydatid may open into the duct and cause great pain, jaundice, and death. As hydatid tumours have been known to form in the kidneys and in the omentum, the presence of a large swelling on the right side need not be necessarily that of an hydatid in the liver.

Treatment.—Several remedies have been suggested in order to destroy the hydatids; such, for instance, as the iodide of potassium, common salt, calomel, etc. Should the tumour press outwardly, surgical interference may be necessary, the cyst being then opened and the contents evacuated. Murchison recommends puncture with a fine trochar, and the drawing off of a portion of the contents, when the hydatids will probably die. Others recommend the partial emptying of the cyst by means of a small trochar or the aspirator, and then injecting it with the tincture of iodine. Passing two electrolytic needles, and allowing a current from a ten-celled battery to traverse the tumour for ten or more minutes, has destroyed the hydatids, a cure being thus effected. After the operation, slight circumscribed peritonitis may occur.

DIAGNOSIS OF HYDATID DISEASE OF THE LIVER.

1. Hydatid tumour of the liver is irregular in its enlargement.
2. In painless diseases of the liver—fatty, lardaceous, or waxy liver, and simple hypertrophy—the enlargement is uniform in all directions; in hydatid, the enlargement is generally most marked in one particular direction—upwards, downwards, or to either side.
3. Hydatid may sometimes be felt as a globular swelling on the surface, as it were, of the liver. Sometimes there are two or more swellings. The right lobe is that most frequently attacked.
4. The tumour may fill the abdomen, push up the lungs, displace the heart, or bulge out the lower ribs on the right side.
5. The tumour is tense, but elastic, and with care is felt to fluctuate.
6. Auscultation may detect, if the walls are thin and the cyst large, a peculiar vibration, *frémissement hydatique.*
7. Movable—moves with the liver; mobility is therefore a valuable diagnostic sign.
8. Ascites, œdema of the lower extremities or hæmorrhoids are rare, as the portal vein is not compressed by the tumour, which points out into the abdominal walls; but the above may occur.
9. Jaundice seldom occurs in hydatid disease: when present it may be due to closure of the common duct from pressure or ulceration.
10. Hydatid tumours grow slowly and without pain.

Table giving the Differential Diagnosis of the following Diseases of the Liver.

HYDATID.	ABSCESS.	CANCER.	ENLARGEMENT OF GALL BLADDER.
Smooth globular tumours, developed slowly, unattended with pain, fever, or marked derangement of general health. Fluctuation or hydatid vibration.	Rapid development, fever, and pain, and early appearance of cachixia.	Uneven surface, density, and tenderness. Cachixia. Do not mistake large soft cancers with a feeling of fluctuation for hydatid.	Most cases preceded by jaundice and colic. Note situation of the tumour.

Note.—Do not mistake aortic aneurism for hepatic tumours. Aneurisms are spindle-shaped, pulsate, and painful. Hydatid rising high in thorax may be mistaken for pleuritic exudations; note outline of dulness, as differing from that of pleuritic effusion. Hydatid dulness is arched upwards, not so pleuritic effusion. Heart more displaced to left in hydatid than pleuritic effusion.

JAUNDICE.

Lat. *Morbus regius.* Fr. *Jaunisse.* Ger. *Gelbsucht.*

Jaundice or icterus is strictly not a disease *per se*, but consists in the collection of symptoms, resulting from the presence of bile in the blood, either from its not being secreted or from some impediment to its excretion and discharge, so that it enters again into the circulation.

Tabular View of the Causes of Jaundice.

A.—*Jaundice from Mechanical Obstruction of the Bile Duct.*

I. Obstruction by Foreign Bodies within the Duct.
   1. Gall-stones and inspissated bile.
   2. Hydatids and distomata.
   3. Foreign bodies from the intestines.

II. Obstruction by Inflammatory Tumefaction of the Duodenum, or of the Lining Membrane of the Duct, with Exudation into its Interior.

III. Obstruction by Stricture or Obliteration of the Duct.
   1. Congenital deficiency of the duct.
   2. Stricture from perihepatitis.
   3. Closure of orifice of duct in consequence of an ulcer in the duodenum.
   4. Stricture from cicatrisation of ulcers in the bile ducts.
   5. Spasmodic stricture.

IV. Obstruction by Tumours closing the Orifice of the Duct, or growing in its Interior.

V. Obstruction by Pressure on the Duct from without, by—
   1. Tumours projecting from the liver itself.
   2. Enlarged glands in the fissure of the liver.
   3. Tumour of the stomach.
   4. Tumour of the pancreas.
   5. Tumour of the kidney.
   6. Postperitoneal or omental tumour.
   7. An abdominal aneurism.
   8. Accumulation of fæces in the bowels.
   9. A pregnant uterus.
   10. Ovarian and uterine tumours.

B.—*Jaundice independent of Mechanical Obstruction of the Bile Duct.*

I. POISONS IN THE BLOOD INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE.

1. The poisons of the various specific fevers.
    (*a*) Yellow fever. (*b*) Remittent and intermittent fevers. (*c*) Relapsing fever. (*d*) Typhus. (*e*) Enteric or pythogenic fever. (*f*) Scarlatina. (*g*) "Epidemic jaundice." Acute Lobar Pneumonia
2. Animal Poisons.
    (*a*) Pyæmia. (*b*) Snake-poison.
3. Mineral Poisons.
    (*a*) Phosphorus. (*b*) Mercury. (*c*) Copper. (*d*) Antimony, etc.
4. Chloroform or ether.
5. Acute atrophy of the liver.
6. Advanced cirrhosis of the Liver -

II. IMPAIRED OR DERANGED INNERVATION INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE.

1. Severe mental emotions, fright, anxiety, etc.
2. Concussion of the brain.

III. DEFICIENT OXYGENATION OF THE BLOOD INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE.

IV. EXCESSIVE SECRETION OF BILE, MORE OF WHICH IS ABSORBED THAN CAN UNDERGO THE NORMAL METAMORPHOSIS.

1. Congestion of the liver.
    (*a*) Mechanical. (*b*) Active. (*c*) Passive.

V. UNDUE ABSORPTION OF BILE INTO THE BLOOD FROM HABITUAL OR PROTRACTED CONSTIPATION.

—*From* MURCHISON.

Symptoms.—The signs which mark ordinary catarrhal jaundice are as follow:—Yellowness of the conjunctivæ and skin, high-coloured urine, due to the presence of the colouring matter of the bile in that fluid. Sometimes in the course of jaundice the urine will become almost normal in colour, whilst the tint of the skin may remain for days unchanged; this may be due to the sudden removal of the cause of obstruction, to the flow of bile, and to the relatively long time required to remove the colouring matter of the bile from the skin. The stools are white, like pipe-clay, and very fœtid, from putrefactive decomposition. The functions of digestion are at first more or less impaired, the patient complaining of depression of spirits and a feeling of general discomfort. The tongue is coated, and the patient complains of a bitter unpleasant taste in the mouth. The breath is unpleasant. The yellowness of the skin now makes its appearance. The pulse may become very slow, 50 to 40 beats a minute. The skin may be hot, and the thirst is in some cases great. In other cases there is no complaint or uneasiness, and I have had patients at the Royal Dispensary who, when asked "What is the matter?" replied "A touch of the jaundice," no other marked symptom being present than the yellowness of the skin. An intolerable itching of the skin not unfrequently annoys the patient. In acute cases the colour of the skin is, as a rule, of a brighter yellow than in those of a chronic nature, when the coloration assumes an olive or greenish-yellow tint. The acute forms are generally attributable to catarrh of the mucous membrane of the gall ducts, which for a time obstructs the passage of the bile. Delirium, when the disease ends fatally, closes the scene.

Diagnosis and Prognosis.—The following method may be adopted to determine whether the jaundice be the result of suppression or obstruction:—Take two or three drachms of the urine and add to them about half a drachm of strong sulphuric acid, so that the two fluids be kept apart as much as possible. Now drop in a piece of lump sugar. If a purple or scarlet line be formed at the line of junction of the two liquids, the case is probably one of obstruction. A slight browning points to suppression. The above test often fails, but we can readily detect the presence of the bile pigments by the following method:—1. Take an inch and a half of urine in a test-tube. 2. Pass slowly down the side of the test-tube a little fuming nitric acid. At the union of the two a play of colours, green, blue, violet-red, etc.

The presence of the green is the only important indication of bile pigment, *bilirubin*. The prognosis will depend upon the probable cause.

Treatment.—In the treatment of jaundice the cause must be looked for, and, if possible, removed. Gentle laxatives—rhubarb, aloes, etc.—to relieve constipation will be required. The diet must be of the simplest—milk, beef-tea, etc. In an adult, if not contra-indicated by the presence of Bright's disease, five grains of calomel, followed by a black draught, will often prove of the greatest advantage. Saline purgatives are most suitable for catarrhal jaundice which supervenes upon gastro-intestinal catarrh. Diuretics should be given to remove the colouring matter of the bile from the blood. The muriate of ammonia, combined with taraxacum, has a most beneficial effect in some forms of chronic jaundice, even after other remedies have failed. The mineral acids combined with bitter infusions are frequently of great use, and in the anæmic condition which often follows an attack of jaundice the salts of iron must be administered. In chronic congestions of the liver with obstinate catarrh of the bile ducts and mucous membrane of the stomach and duodenum, the mineral waters, of Karlsbad, Kissingen, Vichy, Ems, Marienbad, etc., can scarcely be replaced by other remedies for their good effects. Blisters often do good when the inflammatory condition of the liver has somewhat subsided. In those of a gouty diathesis, a few drops of colchicum wine, given with other medicines, will often assist in the success of the treatment. The diet should be simple and easily digestible, and during convalescence gentle exercise should be taken.

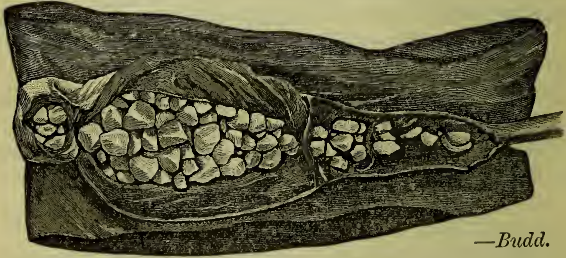
INFLAMMATION OF THE GALL BLADDER.

The gall bladder and its ducts may be attacked by inflammation, giving rise to symptoms of varying intensity, and from which a diagnosis of the cause or causes at work is very difficult. As has been pointed out just now, jaundice may occur from the temporary occlusion of the duct, consequent on inflammation, originating most frequently by the propagation of a catarrhal condition of the stomach and intestines. If this condition continue for any length of time, the gall bladder becomes distended with bile to an alarming extent. Ulceration may take place in the gall bladder, and its contents be emptied into the intestines. Cancer, usually a secondary deposit of that disease, may occur in this organ.

The *Ascaris lumbricoides*, the *Distoma hepaticum* or *Fasciola hepatica* and the *Distoma lanceolatum*, have been known to make their way into the gall duct and create an obstruction to the flow of the bile.

GALL-STONES.

Collections of cholesterine and inspissated bile may take place in the gall bladder. These collections, or "gall-stones" as they are called, may occur singly, but more frequently the gall bladder is found full of them, varying greatly in size; as many as two thousand have been counted after death.



—*Budd.*

Symptoms.—The passage of gall-stones down the biliary duct into the intestine gives rise to important symptoms. Pain, which attends their passage, comes on suddenly without any obvious cause, and while the patient appears in perfect health. It is of a most excruciating character, and is felt in the right hypochondrium. The paroxysm varies considerably as to its duration, often terminating suddenly and followed by comparative ease, some slight tenderness only being felt in the part which a few minutes before was the centre of intense pain. After a few days the gall-stone may be found in the evacuations. One attack may be followed by many more.

Treatment.—The treatment consists in the administration of opium to relieve the pain, but care should be taken not to give too much, as the sudden cessation of the pain permits the full action of the drug. As sedentary habits tend to their formation, a more active life should be enjoined. Horse exercise should, if possible, be indulged in.

DISEASES OF THE PANCREAS.

So little is known concerning the diseases to which the pancreas is liable, that a passing notice is all that is necessary. The gland may be the seat of acute or chronic inflammation and of cancer. Calculi may be formed in the pancreatic duct which may give rise to obstruction of the flow of the pancreatic secretion, or to dilatation of the duct. The diagnosis will depend upon the freedom of neighbouring organs from disease, and attendant symptoms.

DISEASES OF THE SPLEEN.

Like other organs, the spleen may become the seat of acute and chronic inflammation. Primary or secondary cancer of the spleen is very rare. The spleen may also be the seat of tubercle, which may occur in two forms. First, as extremely small grey granulations, varying in size from a peppercorn to a millet seed. By washing the spleen in a stream of water, the malpighian bodies are washed away, leaving the tuberculous matter behind. The other form occurs in larger masses.

As an aid to the diagnosis of their true nature, the following points should be remembered:—

(*a*) "A generally rounded figure of the masses, which vary in size."

(*b*) "An almost complete caseous transformation of the deposit."

(*c*) "The presence of miliary-sized granulations forming the recent outer edge of the patch, which, if large, may be softening down in the centre."

Structural lesions may also occur in the spleen. Chronic enlargement, readily detected by percussion and palpation, is seldom attended with pain. This enlargement may be associated with intermittent fever "ague cake" —leucocythemia and cirrhosis. The spleen has been removed by surgical operation, when the enlargement has been very great, but always with a fatal result.

LARDACEOUS OR WAXY SPLEEN.

The spleen is frequently affected by lardaceous or waxy degeneration. The amyloid matter may be diffused throughout the gland, but in most cases the malpighian corpuscles are the parts most affected. These become greyish and semi-pellucid; not unlike sago grains, embedded in the stroma of the organ, hence the name "sago spleen." The application of iodine to the cut surface of the gland produces the brown colour characteristic of the reaction of this substance on the lardaceous or amyloid material. In this disease, the parenchyma, as well as the small arteries of the malpighian capsules, are implicated. The organ is not, as a rule, enlarged in "sago spleen," but in the ordinary form of lardaceous disease the gland is enlarged, anæmic, and of a homogeneous appearance, with a peculiar translucent and lustrous look on section. To the touch it has the feel of firm lard or raw bacon fat.

LEUCOCYTHÆMIA.

Lat. *Leucocythæmia.* Fr. *Leucocythémie.* Ger. *Leukämie.* Syn. *Leucocythæmie.*

Definition.—A morbid combination of the blood, with increase in the white corpuscles of the blood and of lymphatic tissue in one or more of the internal organs.

Leucocythæmia, or Leukæmia, is a morbid condition of the blood, in which the white corpuscles are abnormally increased. In health the proportion of white corpuscles to red is about one in three hundred; but in leucocythæmia the proportion is one in twenty, or even less. This state of the blood is very generally associated, first, with enlargement of the spleen, and the lymphatic glands of the neck, axilla, and groin (due to the formation of lymphatic tissue in them), the tonsils, follicular structures of the intestines, medullary tissue of bones, liver, kidneys, lungs, etc., being secondarily attacked. The spleen becomes first engorged with blood, and then the cells of the pulp are increased in number, the malpighian follicles being but slightly affected. At a later stage induration from hypertrophy of the fibrous elements of the gland takes place, accompanied with exudation, and masses resembling blocks or infarcations are formed, which sometimes undergo degeneration. Enlargement of the cortical portion and multiplication of their cellular elements predominates in the lymphatic glands. Secondary morbid changes, commonly found in the mucous membrane of the intestines, now occur, which take the form of tumours or growths, composed chiefly of lymph cells and a more or less clearly developed stroma. Besides these growths diffuse infiltrations of a like nature may be found, showing that the morbid change begins in all organs in the interstitial connective tissue or stroma and not in the glandular or secretive elements. These infiltrations are held by some authorities to be due to the migration of the leucocytes from the blood-vessels; others maintain that they result from proliferation of the corpuscles of the connective tissue. In the retina minute lymphatic formations, with paleness of the vessels and peculiar white spots—*Retinitis leukæmica*—may be seen with the ophthalmoscope.

The investigations of Bennett, Virchow, etc., have shown that there is an intimate connection between leucocythæmia and changes in the spleen, lymphatic glands, and the red medulla of bones which are believed to take part in the formation of the colourless blood corpuscles. Hypertrophy of these structures causes an increase of corpuscles, but not in all cases, for hypertrophy may occur without causing leucocythæmia. Four forms of leucocythæmia have been described—the splenic, the lymphatic, that due to changes in the osseous medulla, and the fourth to hypertrophy of the glandular structures of the intestines. In the splenic form the corpuscles are said to be somewhat larger than the red, which they closely resemble, but frequently containing multiple nuclei; in the pure lymphatic form the corpuscles are slightly smaller than ordinary white blood corpuscles, but with larger nuclei. The exact cause of this disease has not, however, been clearly made out; but syphilis, ague, and in women exhaustion after parturition, have all been put forward as probable causes.

Symptoms.—The disease, as a rule, commences insidiously, the symptoms which accompany leucocythæmia being chiefly those of anæmia, with great weakness and extreme pallor. Anorexia is often present, and passive hæmorrhages frequently occur, together with œdema of the legs and enlargement of the abdomen. The abdomen becomes enlarged, due probably to the increased size of the liver, spleen, and lymphatic glands of the abdomen. Leucocythæmia is more frequent in men than women, and more common in adult life than in youth, and most in advanced life. No case of recovery is known.

Treatment.—Tonics, quinine, iron, cod-liver oil, iodide of potassium, and general measures for improving the health.

HODGKIN'S DISEASE OR LYMPHO-SARCOMA, MALIGNANT LYMPHOMA, LYMPHADENOMA.

This disease is characterised by the development of lymphatic growths in the spleen, liver, and other organs similar to those occurring in leucocythæmia, from which it differs, however, in there not being that marked increase in the white blood cells, which is so marked in the last-named disease.

The spleen is "increased more or less in size, and contains a number of deposits of yellowish-white, tolerably firm opaque matter, resembling at first glance masses of suet scattered through the organ, or more rarely collected into a larger mass in it. The masses are generally of angular outline of very various sizes, and embedded deeply in the spleen tissue."

Treatment.—Arsenic may be tried in this disease, but in most cases all kinds of treatment have proved useless.

DISEASES OF THE NERVOUS SYSTEM.

INFLAMMATION OF THE BRAIN.

Inflammation occurring within the cavity of the head may attack the membranes or the substance of the brain. The inflammation may be either *acute* or *chronic.*

ENCEPHALITIS.—*Definition*—"Inflammation of the brain or of its membranes." This term is to be used only when the precise seat of the inflammation has not been ascertained by *post-mortem* examination.

MENINGITIS.—*Definition*—Inflammation of the membranes of the brain.

1. Inflammation of the dura mater.—"This form of inflammation is almost invariably the result of injury or disease of the bones of the skull."
2. Inflammation of the pia mater and arachnoid occurs in the course of the exanthemata.

INFLAMMATION OF THE BRAIN.

Lat. *Inflammatio cerebri.* Fr. *Cérébrite.* Ger. *Entzündung der Hirnsubstanz.*

Definition.—Inflammation of the brain substance, with or without implication of the membranes, usually partial, and in many cases dependent on local injury or foreign deposit.

For all practical purposes the distinction between meningitis and cerebritis is unnecessary, as the one seldom occurs without the other.

But as the student may be required to show his ingenuity in diagnosis, the following Table, taken from Aitken, may perhaps be of some assistance:—

CEREBRAL DISEASE.	MENINGEAL DISEASE.
1. From the outset, or from a very early stage of development, there is loss of some one or more of the proper nervous functions, shown by paralysis, anæsthesia, loss of memory.	1. It is not till some time after the detection of signs of disease, that diminution or loss of nervous function takes place.
2. Cerebral disease is not commonly attended with highly-marked exaggeration of function, such as furious delirium, convulsions, intense hyperæsthesia, pain, or tenderness.	2. The subsequent diminution or loss of nervous function which succeeds the prolonged existence of "head symptoms" is generally preceded in cases of meningeal disease, by extremely severe excitement, or exaggeration of functions, such as pain, tenderness, furious delirium, or convulsions.
3. Little vascular excitement attends cerebral disease, nor is there frequently any highly-marked general disturbance.	3. In meningeal affections there is usually much local vascular excitement, with general disturbance.
4. Paralysis and anæsthesia, losses of volition, ideation, perception, and the like, characterise cerebral disease.	4. Spasms, convulsions, pain, and delirium, are the general features of meningeal disease.

Pathology.—Acute inflammation attacking the membranes of the brain is attended with the same results as when it occurs in other serous membranes. There is more or less vascular redness due to engorgement of the vessels, and in most cases effusion of serum or deposit of lymph takes place. When the effusion is between the arachnoid surfaces, it is probably the result of disease of the dura mater, the result of injury; but if the effusion be found between the arachnoid and the brain, it is not due to violence, and is, therefore, the result of idiopathic inflammation of the membranes. In simple meningitis the space beneath the visceral arachnoid is covered with lymph, frequently of a greenish colour, and purulent. Apart from mechanical injury, it is not always possible to assign the cause of this disease. It may occur during the course of any of the fevers, during an attack of gout, rheumatism, erysipelas of the head, lengthened exposure to the sun, excessive mental strain, or it may be due to habits of intemperance. Suppression of the menses is, according to Dr Abercrombie, a frequent cause, especially in weak, unhealthy young women. Inflammatory affections of the brain are more common in early childhood and in young adults than during any other period of life.

Fatal meningitis very frequently follows a chronic discharge from the ear. A distressing case occurred a few years ago in Edinburgh. The son of a medical man, a fourth-year's student, was suddenly attacked by this disease. He attended a midwifery case the night before the pain in the head began. A discharge of pus from the ear on the second day gave a slight but temporary relief, and he died within a week. Pus was found on the base of the brain, and the petrous portion of the temporal bone was diseased.

Symptoms.—Usually the earliest symptoms of inflammation of the brain and its membranes are severe pain in the head and giddiness, followed by repeated rigors and vomiting, accompanied with more or less pyrexia. In children an attack of convulsions is not unfrequently the first symptom to attract attention.

During the *first stage* the head is hot, the face is flushed, and the carotids and temporal arteries pulsate strongly. The eyes are bloodshot and watery, and there is great intolerance of light, and often there is marked squinting. The bowels are, as a rule, obstinately confined. The pulse is quick, strong, and full. Delirium, more or less active, occurs in this stage, which, if the inflammatory action be not early arrested, passes on in a few hours, or at most three days, to the second stage, in which there is gradual and persistent depression of the functions of the brain, marked by relaxation of the muscles, immobility, and dilatation of the pupils and coma. Niemeyer remarks that "fever of similar character and equal severity occurs in scarcely any other disease of the brain, and consequently is very important in the diagnosis of meningitis. If the frequency of the pulse disappear after the disease has lasted for some time—if it fall from 120–140 beats per minute to 60–80 beats, while the other symptoms of fever and the functional disturbances of the brain increase—the evidence is still more in favour of meningitis."

In the *second stage*, the pain in the head, fever, and delirium diminish, and there is less sensitiveness to light and sound. The pupils, in the first stage contracted or oscillating, now become dilated; an unpleasant squint is present, accompanied with constant restlessness of the eyeballs. The patient, in fact, is gradually getting comatose. The pulse becomes slow and irregular, and the respirations laboured. Sordes form round the sockets of the teeth, and the sphincters are relaxed, so that the patient passes his motions as he lies in bed. Convulsions may occur, sometimes just before the fatal termination.

The following Table will show the characteristic differences between gastric or hepatic vomiting and that due to cerebral disease:—

GASTRIC OR HEPATIC VOMITING.	CEREBRAL VOMITING.
1. There is nausea, relieved, at all events temporarily, by the discharge of the contents of the stomach.	1. Nausea absent or very slight. Vomiting continues in spite of the stomach being empty. Any fluid or solid taken is immediately rejected.
2. The tongue is coated, the breath foul, the conjunctivæ yellowish, and the *headache secondary* in point of time.	2. The tongue clean, the breath pure, the conjunctivæ colourless or only injected, and the *headache primary*.
3. Vomit consists of undigested food, bile, and offensive secretions ; sometimes acid water, pus, or blood.	3. Vomit consists of unaltered food, frothy mucus, never pus or blood, and sometimes only a trace of bile.
4. Griping pain in abdomen, diarrhœa, fœtid eructations, and unhealthy watery stools.	4. Obstinate constipation, solid healthy stools, and no unpleasant eructations.
5. Loss of appetite.	5. Appetite present, no disinclination to take food.
6. Retching and increased salivation, abdominal tenderness, faintness, and exhaustion.	6. The stomach is emptied almost without effort; no increase of the saliva; slight, if any, abdominal tenderness; no feeling of faintness after vomiting.

When nausea and vomiting are the initial symptoms, the inflammation is considered by some to have its origin in the substance of the brain; but when convulsions begin the attack, the membranes are thought to be the first implicated. So, also, if the inflammation be local, the symptoms become limited. Sub-orbital or sub-occipital pain, with paralysis of parts supplied by the cranial nerves, pointing to the base of the brain as the seat of the disease. In these cases the mental and the sensorial excitement are less pronounced.

Diagnosis.—Acute inflammation of the brain or its membranes when attended with active delirium must not be confounded with the delirium which occurs in the course of typhoid fever. The history of the case will help to form a correct diagnosis. The slow development of typhoid, the presence of diarrhœa, tympanites, tenderness of the iliac regions, and continuance of the delirium without the supervention of coma, will mark the distinction between them. From *delirium tremens*, the busy delirium, the half-sensible, half-senseless loquacity of the patient, who can be aroused from time to time to reply to questions in a rational manner, will, together with the trembling, half-cowardly actions of the sufferer, help to form our diagnosis of the case.

Treatment.—The indications for treatment are—

(*a*) To relieve the congestion of the brain, and arrest the inflammatory action.

(*b*) To sustain the strength of the patient, and promote absorption of the effused fluid.

If the general condition of the patient do not contra-indicate it, local or general bleeding may be necessary. The hair should be shaved off, and ice in bladders applied to the head. Active purgation is in most cases called for. Perfect quietness must be enjoined, and everything that tends to increase the cerebral excitement must be avoided. The patient should be kept scrupulously clean, and tepid sponging will be found very grateful to him. The diet in the first stage should be bland and unstimulating. When, however, the powers are failing, and fatal collapse appears imminent, recourse must be had to stimulants—strong beef-tea, ammonia, wine, or even brandy. To remove the effusion, blisters to the back of the neck and the inunction of mercurial ointment on the scalp, together with the internal exhibition of the iodide of potassium, are the measures to be adopted. A catheter should be passed at regular intervals into the bladder, if the patient be unable to pass his water. It should be remembered that the application of cold to the head has a most depressing effect; the case should therefore be most carefully watched when this method of treatment is adopted.

CHRONIC INFLAMMATION OF THE BRAIN.

Chronic Inflammation of the Brain, not due to tubercular deposit, may come on independently of a previous acute attack. The symptoms which sometimes accompany this disease are often not calculated to draw the attention to the brain, and not unfrequently a case of chronic meningitis has been treated as a case of chronic gastritis. Pain in the head, together with much gastric irritation, are often prominent symptoms. Taken generally, the symptoms are those of incipient insanity. There is more or less mental apathy, and a disinclination to exertion. The general health becomes impaired, and there may be partial paralysis of some parts of the body, and perhaps at the same time rigid contraction of the muscles in others. Delirium, or even fits of mania, may supervene, and the patient dies in an asylum during an attack of convulsions followed by coma. After death, if the brain be examined, the membranes may be found more or less thickened, and covered here and there by deposits of lymph. The structure of the brain is softened—*ramollissement*—and may be almost deffluent, having the appearance of raspberries and cream. In some cases an abscess is formed, and this is especially likely to occur when there is disease of the petrous portion of the temporal bone.

The diagnosis of chronic abscess from tumours of the brain is by no means easy. A history of injury followed by incephalitis, and then a period of two or three months of obscure symptoms, points to abscess; if the uncertain symptoms prevail for a much longer period, the probability is on the side of tumour. Double optic neuritis, preceded by vomiting and headache, points to tumour of the brain.

The treatment must be regulated by the condition of the patient, chiefly consisting in quietude of mind and body, and improving, if possible, the general health.

TUBERCULAR MENINGITIS.—ACUTE HYDROCEPHALUS.

Lat. *Meningitis tuberculosa.* Fr. *Méningite tuberculeuse.*
Ger. *Tuberculöse Hirnhautentzündung.*

Definition.—Tubercular meningitis is an inflammatory affection of the membranes of the brain, accompanied with the deposit of miliary or yellow tubercle on or between the membranes at the base of the brain. The disease occurs in young scrofulous or strumous children; and, besides the condition just mentioned, the head increases in size, the child wastes and dies from the brain mischief or from some interconcurrent disease. The disease may occur in the adult during phthisis.

Tubercular meningitis differs from simple meningitis in the presence of tubercular granulations at the base—in rare cases on the convexity—of the brain. The tubercles are probably deposited before the inflammatory exudation takes place, and they will be found following the course of the small arteries and situated between the vessel and its perivascular sheath, sometimes by their pressure reducing the calibre of the artery. On carefully separating the fissure of Silvius, their presence will be more surely made out. The inflammatory action is in the majority of cases situated at the base of the brain, in this respect differing from the simple forms of meningitis. The morbid appearances are increased vascularity of the membranes and cerebral structure, together with an effusion of turbid fluid under the arachnoid and into the ventricles. Flattening of the convolutions will more or less depend on the quantity of the effused fluid. The sides of the ventricle are soft, and the fornix, septum lucidum, etc., are often reduced to mere shreds. The softening is probably the result of inflammatory œdema; some observers, however, maintaining that it is the result of the fluid in the ventricles, the parts becoming soddened. This disease is generally accompanied with the deposit of tubercle in other organs of the body, and in adults is not unfrequent in the course of phthisis.

Tubercular meningitis was formerly known and described as acute hydrocephalus. Some writers, under the term *Leptomeningitis Infantum*, describe a form of acute hydrocephalus occurring in children but *not of tubercular origin.* These writers hold that the disease does not always arise from inflammatory action.

The diagnosis of the two diseases is by no means easy, the premonitory signs and symptoms of the tubercular being generally absent in the non-tubercular variety. Recovery is also more frequent in the latter than the former.

The symptoms and treatment do not differ in any essential way from the disease under consideration.

TABLE SHOWING THE POINTS OF DIFFERENCE BETWEEN TUBERCULAR AND SIMPLE MENINGITIS.

TUBERCULAR MENINGITIS.	SIMPLE MENINGITIS.
1. Presence of lymph, as a rule, at *base* of brain.	1. Presence of lymph on *surface* of brain.
2. Flattening of the hemispheres.	2. Absence of flattening.
3. Tubercles in the pia mater following the course of the vessels, and in other parts of the body.	3. Absence of tubercles in the brain and in other parts of the body.
4. Greatly increased amount of fluid in the ventricles (three to four ounces), and softening of their walls.	4. Fluid but slightly increased and absence of softening.
5. Occurs most frequently in scrofulous or tuberculous children.	5. Occurs during, or as a result of, the exanthemata, scarlet fever, measles, etc.

Symptoms.—The early symptoms of tubercular meningitis in the child are those which mark a general impairment in the health of the individual. The appetite becomes capricious, the temper irritable; there is a diminution in the weight of the body, and an anæmic condition of the surface. The child may be drowsy, yet sleeps badly, the eyes being frequently half open. Sooner or later the head symptoms make their appearance, there is pain in the head and giddiness, the child "suddenly standing still, gazing round for a moment as if lost, then, either beginning to cry at the strange sensation, or seeming to awake from its reverie and at once returning to its play." Then follow vomiting, increased sensibility to light and sound, sudden and frequent flushing of the face, often the forerunner of convulsions. In young children the fontanelle bulges out and throbs. Optic neuritis is seldom present, but, in cases of general miliary tuberculosis tubercles may be seen in the choroid. The bowels are confined and the tongue furred.

Sometimes, due to the presence of tuberculous ulceration of the intestines, diarrhœa, instead of constipation, is present, and may lead to an error in diagnosis—typhoid fever,—unless the chance of this complication being present be remembered. The irregularity of the respirations Trosseau considered as pathonomonic of the disease. The pain in the head may not be constant, but may occur at intervals, and is of a lancinating nature, giving rise in infants to a short, sudden, sharp cry, known as the *cephalic cry.* When the child is old enough to speak, he often exclaims, "Oh! my head," at the same time lifting his hands to his head.

As the disease progresses, drowsiness and other signs of cerebral compression set in, the pulse becomes slow and irregular, and the respirations are performed with difficulty, and often attended with sighing. The child often grinds his teeth together and moans in a most painful manner, and its little hands are constantly used to pick its nose and lips till they bleed. Gradually the little patient becomes worse, and dies either in convulsions or comatose.

Tubercular meningitis must be discriminated from infantile remittent fever. The diagnosis will be assisted by the absence, in infantile fever, of a morbid sensibility to light, and of the peculiar darting pain in the head, so frequent a symptom of meningitis. Squinting and rolling of the eyeballs, in the last stage of the latter disease, will help to distinguish the one from the other.

The disease must also be distinguished from cerebrospinal meningitis, by the absence in the former of a *severe initial chill*, pain in the neck and back, etc.; and from tumour of the brain, by the absence of the well marked double optic neuritis in tumour, and by the suddenness of the symptoms which mark the onset of tubercular disease.

In simple gastric disorder, the vomiting and the confined bowels are soon relieved by a brisk purge, the child rapidly recovering after the bowels have been well relieved.

From ear-ache it may be distinguished by the pain in the latter being on one side, relieved by warmth, and by the appearance of redness in the meatus, and sometimes of a fœtid discharge, followed by almost immediate relief.

The symptoms in the early stages of pneumonia may be mistaken for tubercular meningitis. In pneumonia, the vomiting, though violent at first, soon ceases, and leaves no irritability of the stomach; the alvine discharges are natural; the heat of the skin, more marked about the body than the head, is less than in tubercular meningitis, and the physical lung signs are present.

Many mistakes may be avoided by examining every organ in rotation. In pleurisy, the physical signs of that disease are present.

TABLE SHOWING THE POINTS OF DIFFERENCE BETWEEN TUBERCULAR MENINGITIS AND THE REMITTENT OR TYPHOID FEVER OF CHILDREN.

TUBERCULAR MENINGITIS.	REMITTENT FEVER.
1. Nearly half the cases of this disease occur under five.	1. Rare under five, and seldom met with under three.
2. Vomiting present from the first, followed by persistent nausea.	2. Vomiting in most cases absent, even at onset of fever, and if present soon ceases.
3. Bowels confined; motions scanty, dark, or mud-coloured.	3. Bowels relaxed, watery, lightish in colour, and fæcal. Tenderness over abdomen, especially in iliac regions.
4. Tongue moist, and covered with a thin white fur.	4. Tongue dry, furred down centre, red at tip and edges.
5. Frequently a distaste for drink and food.	5. Constant desire for drink, but distaste for food.
6. Dryness of the surface, but temperature not much raised.	6. The skin is very hot.
7. Delirium not an early symptom, complains constantly of pain in the head.	7. Delirium is of early occurrence; but slight complaint as to head.
8. No definite remissions, although many indefinite fluctuations.	8. Symptoms remit; better in the morning, worse at night, and so on.

TABLE GIVING THE DIFFERENTIAL DIAGNOSIS OF THE FOLLOWING DISEASES.

TUBERCULAR MENINGITIS.	CEREBRO-SPINAL MENINGITIS.	TYPHOID FEVER.	'HYDROCEPHALOID' DISEASE.	SIMPLE MENINGITIS, 'BASILAR OR CONVEX.'
Acute invasion, absence of double optic neuritis, history of case, prodromata.	Pain in nape of neck and back. Absence of *severe initial chill.*	Temperature peculiar to typhoid, tympanitic abdomen, diarrhœa, enlarged spleen.	Extreme depression, quiet somnolence, depressed fontanelle.	Very sudden attack, no premonitory symptoms, higher fever, more acute delirium than in the tubercular.

NOTE.—Tubercular disease may simulate delirium tremens, hysteria, abscess, hyperæmia, thrombosis of cerebral sinuses, and leptomeningitis infantum.

In the *adult*, during the course of phthisis, tubercular meningitis not unfrequently supervenes, and as the symptoms of the latter may be at first obscure, care is required in the diagnosis. Thus a phthisical patient may appear wayward, petulant, and even obstinate, and unless the possibility of the presence of tubercular meningitis be remembered, grave errors may be committed. The head symptoms, however, generally come on suddenly without any premonitory warning, the first and prominent symptom being violent pain in the head. Amelioration of the chest disease for a time occurs in most cases. Vomiting, slight fever, and an irregular feeble pulse soon follow.

In some cases, the early symptoms may be those of an apoplectic fit, followed by convulsions. The patient soon becomes worse, the mental dulness becomes greater, or there may be some amount of delirium. As the disease proceeds the sphincters relax, the stupor increases, and he dies in about a fortnight or less, death sometimes being preceded by a remission of the symptoms.

The *post-mortem* reveals the presence of tuberculous deposits on the membranes at the base of the brain, varying in size from that of a millet seed to that of a pea. Sometimes there is a layer of yellowish opaque exudation, particularly in the neighbourhood of the optic chasm and the fissure of Silvius.

There is no symptom peculiar to basilar meningitis which will alone render its diagnosis certain; but the previous phthisical history will point to this form of meningitis.

Treatment.—The treatment in the case of the child will consist in the use of purgatives at the outset, followed by the administration of the iodide of potassium at regular intervals. Ice should be applied to the head, and local bleeding by leeches may be had recourse to if the symptoms demand its use. Chloral may be given either by the mouth or rectum.

In the adult the treatment is more or less expectant; the patient generally dies either of the pulmonary affection, or is killed outright by the severity of the brain lesion.

CHRONIC HYDROCEPHALUS.

Lat. *Hydrocephalus longus.* Fr. *Hydrocéphale chronique.*
Ger. *Chronischer Hydrocephalus.*

DEFINITION.—A disease of infancy and early childhood, probably of inflammatory origin, accompanied with increase of fluid in the ventricles of the brain, enlargement of the head and general emaciation. The child generally dies from mischief to the brain or from some interconcurrent disease, measles, etc.

Chronic Hydrocephalus, or Dropsy of the Brain, is sometimes congenital, and then is usually associated with some malformation of the brain. When not congenital, the causes of the disease are obscure. Some consider the disease to be the result of an arrest of the development of the brain.

Dr West, however, is of opinion "that the disease is not a mere passive dropsy, nor simply a consequence of arrested cerebral development, but that it is the result of a slow kind of inflammation of the arachnoid, especially of that lining the ventricles, which may have existed during fœtal life, or may not have attacked the child until after its birth. I may further add that each year leads me to estimate more highly the share of inflammation of the lining of the ventricles in the production of chronic hydrocephalus."

Symptoms.—Among the early symptoms of this disease may be noticed an impairment in the general health, signs of mal-nutrition being also evident. Should the disease be congenital, these are generally present from the child's birth. The child may refuse food, or it may take it readily, but without appearing to be nourished by the amount consumed. The bowels are usually confined, and the evacuations are almost always of an unhealthy character. The head becomes *hot*, and there is always more or less *tension* and *pulsation felt over the anterior fontanelle.* This condition of the fontanelle often attracts attention long before there is any notable increase in the child's head. Sooner or later the head begins to increase in size, and to assume the characteristic hydrocephalic appearance. The child becomes restless and peevish, slight convulsive attacks often occurring without any definite assignable cause. Suddenly the urgent symptoms are arrested, and the little patient gives hope of ultimate recovery, when as suddenly dangerous symptoms again make their appearance, and the child dies in terrible convulsions.

"Few objects," writes Dr West, "are more pitiable than a little child who is the subject of far-advanced chronic hydrocephalus. While the skin hangs in wrinkles on its attenuated limbs, the enlarged head appears full, almost to bursting, owing to the stretching of the scalp; and the scanty growth of hair does not at all conceal the distended veins that run over its whole surface. The size of the skull, too, appears greater than it really is, since the face not only does not partake of the enlargement, but retains its infantile dimensions much longer than natural. The eyes are so displaced by the altered direction of the orbitar plates that the white sclerotica projects below the upper lid, and the iris is more than half hidden beneath the lower. Often, too, there is a considerable degree of convergent strabismus, or a constant rolling movement of the eyeball, which the child is unable to control; or the pupil is dilated, and quite insensible to light."

Chronic hydrocephalus is in the majority of cases a fatal disease. If the child do not die of the disease itself, it is readily carried off by other ailments not usually fatal in themselves; the end in most cases being ushered in by convulsions or other indications of cerebral mischief.

Chronic hydrocephalus has been divided into two kinds:—

(*a*) EXTERNAL HYDROCEPHALUS.—When the fluid collects in the sac of the arachnoid.

1. Fluid may escape into the cavity of the cranium from yielding of the commissures of the distended brain.
2. Hæmorrhage.
3. Atrophy of the brain—fluid taking the place of the diminished brain.

(*b*) INTERNAL HYDROCEPHALUS.—When the fluid accumulates in the ventricles of the brain.

Treatment.—In most cases the treatment of this terrible disease is only palliative, attempts being made to "ease the stormy passage to the grave."

Professor Golis, of Vienna, has recommended the inunction of the mild mercurial ointment to the scalp previously shaved of the hair. To prevent chills, a flannel cap is to be ordered to be worn; a $\frac{1}{4}$ gr. of calomel is also directed to be given twice a-day. The iodide of potassium or the syrup of the iodide of iron may also be given. Bandaging the head with strips of diachylon plaster has been used by Barnard, Trousseau, and others; and lastly tapping of the head in the line of the coronal suture, about an inch or an inch and a half from the anterior fontanelle, has been practised with variable success, but this should only be resorted to when there is every probability that the case is one of external hydrocephalus.

Whichever plan be adopted to remove the fluid, it must be borne in mind that special care will be required to preserve the strength and improve the health of the little patient.

APOPLEXY.

Definition—Apoplexy is a state of insensibility, passing in some cases, more or less suddenly, into fatal coma, and dependent on several pathological conditions.

Four varieties of apoplexy have been described:—

(*a*) Hæmorrhagic or Sanguineous Apoplexy.—This is caused by the extravasation of blood in the substance of the brain. The most usual seats of the extravasation, taken in the order of their occurrence, are the *corpus striatum* and *optic thalamus;* then the *grey matter* of the *convolutions*, the *cerebellum* and *pons varolii.*

(*b*) Serous Apoplexy.—This is due to the presence of serous effusion into the ventricles, or into the arachnoid cavity.

(*c*) Congestive Apoplexy.—This is brought on by an over-loaded condition of the vessels of the brain.

(*d*) Simple or Nervous Apoplexy.—Cases of simple or nervous apoplexy have been described by Abercrombie, Louis, Andral, and others, where after death there was no appreciable pathological condition to account for the symptoms. These cases are probably due to uræmic poisoning. During infancy, a form of apoplexy known as *meningeal* or *peripheral apoplexy* is not unfrequent.

(*a*) The *predisposing causes* of apoplexy are—

1. Excessive Plethora.—A tendency exists in some persons to accumulate blood rapidly, this tendency being often hereditary. There is also a tendency in some persons to degenerations in the arteries of the brain, and the formation of miliary aneurisms described by Charcot, and held to be a form of arterial sclerosis. Apoplexy is more common between fifty and sixty years of age, when the powers of nutrition are still good, but the power to resist the wear and tear of the system is diminished; it may also occur in women soon after the cessation of the menses. Men are more frequently attacked than women. Children are more liable to meningeal hæmorrhages than those advanced in life.

2. Disease of the Arteries of the Brain.—The affection of the arteries most commonly found in persons about the middle of life, is an atheromatous condition of the vessels, followed by a calcareous deposit in their coats. The atheromatous change is a fatty degeneration or a semi-cartilaginous thickening affecting the inner coats of the arteries, the result of a low form of inflammation, and to which the Germans have applied the term *endarteritis deformans*. The atheromatous patch situated between the inner and middle coats becomes chemically changed into cholesterine; this bursting through the weakened inner coat allows the blood to flow in, causing a dissecting aneurism, stopping the flow of blood through the artery, and causing softening of the brain; or the aneurism may burst, and sudden death from apoplexy result. Sometimes the patch becomes infiltrated with lime salts, and "ossification," so-called, takes place. This is not a true ossification, but merely a deposit of earthy salts in a fibrous matrix. The inner surface is rough, and causes the blood to be arrested in its course with the formation of a clot, which may become dislodged and plug a smaller vessel. The "miliary" aneurisms of Charcot and Bouchard are probably dissecting aneurisms on small arteries produced as above described. If the morbid action have once commenced in the vessels of the brain, it continues to proceed through the rest of life, and hence it is that many emaciated feeble old persons die of apoplexy. Sometimes an atheromatous patch in an artery may lead to its bursting without the intervention of an aneurism. Among the other predisposing causes of apoplexy is renal disease, especially that form known as granular contracted kidney. Here we have several forces at work —hypertrophy without dilatation of the left ventricle, and disease of the arteries. The force of the heart's action is broken when dilatation accompanies hypertrophy; but, in this case, dilatation—the safety-valve,

so to speak—is absent, and the undiminished force of the heart ruptures the diseased arteries. Syphilis may add its quota as a predisposing cause of this disease, as may also hereditary predisposition. One or more of the following factors may be present:—Gout, granular kidney, hypertrophy of left ventricle, and disease of the arteries.

(*b*) *Exciting Causes.*—The exciting causes of apoplexy are over-exertion, fits of ungovernable passion, constipation, hot and ill-ventilated rooms, sudden chills, and the indulgence in too hearty a meal.

Symptoms.—In many cases there is a gradual failure of the memory, particularly of the names of persons and things pointing to mal-nutrition of the brain, but in most cases an attack of apoplexy comes on suddenly, the patient falling, entirely bereft of consciousness, and in a comatose state. Sometimes, however, he may be first seized by some minor symptom, such as giddiness, or a violent pain in the head, when, after the lapse of a few hours, partial paralysis of one arm or one side of the face may supervene. At other times the onset may assume an epileptiform character, when the ultimately paralysed limbs are those which have been previously convulsed. Again, he may only complain of a feeling of intense drowsiness, and then, on being carried to bed, after an hour or two, become completely comatose.

In the last case, if the patient be seen in the early stage whilst he is still able to recognise those around him, and attempt to speak, he will be found in a fainting condition, the surface cold and clammy, and his pulse quick and small from the fright and shock to the ganglionic system. When the state of coma is established, his pulse will become full, slow, and labouring —fifty or less beats in a minute. The surface of the body now becomes preternaturally hot and bathed in perspiration; the breathing is stertorous, owing to the paralysis of the muscles which compose the soft palate;

the countenance has a peculiar bloated appearance, and the lips are drawn in and forced out at each act of inspiration and expiration. The sphincter ani is frequently paralysed, allowing the escape of the contents of the lower bowel, whilst the urine is retained in the bladder. The extremities of one side are generally found more or less paralysed; the pupils of the eyes may be dilated or contracted, or in some cases they may be unequally dilated, and incapable of receiving any impression from light or the finger placed before them. The state of coma may continue for some hours, and then the patient gradually recovers to find one or more of his limbs useless. On the other hand, the patient may die outright in a few minutes, the mode of dying being by apnœa. Should, however, the state of coma continue for some days, he may die from exhaustion and apnœa combined. It must be borne in mind, however, that between the onset of the attack and death or comparative recovery the patient may go through a variety of phases, thus he may be delirious or alternately comatose or semi-conscious. At other times one or more of the affected limbs become rigid.

The author once attended a thin spare lady for apoplexy, who, on recovery, became "stone blind." On consulting an ophthalmic surgeon for her blindness she was recommended the use of "stout" daily, and on this treatment she ultimately recovered her sight. The blindness was due to the rupture of a small vessel in the retina. Two years and a half after the first attack she died suddenly during a second fit of apoplexy.

Post-mortem Appearances.—The presence of a dark red clot in the ventricles of the brain, together with a red coloration of the surrounding tissues, are the *post-mortem* appearances found, should death occur in three or four days. In cases which end more suddenly, there is probably some disease of the heart as well. If the case do not end suddenly, certain reparative changes commence. The serum is rapidly absorbed. The clot becomes decolorised—a pale buff colour on the surface, darker internally. Gradually it disappears, and the uninjured vessels appear across the cavity as fine threads; and as time progresses the clot disappears, and a few "hæmatoidin crystals and grains remain, clinging in the cicatrix." "All this while the injured texture, forming a fibrous scar tissue, closes around the clot, and this may develop well, and produce a cyst-like fibrous sac, the 'apoplectic cyst.' When the clot is gone, this cyst remains filled with clear fluid, crossed, as we stated, by threads" *(Wilks).* Softening of the brain, due to diseased state of the vessels, may be also present. Niemeyer considers that an abscess may form at the seat of an apoplexy; this is considered most improbable by Wilks and Moxon. Sometimes three or more apoplectic patches may be found in different stages of spontaneous cure. Aneurisms, varying in size from a pea to a bean, not unfrequently occur in young people under twenty-one. They affect the larger arteries of the brain, may burst, and then give rise to sanguineous apoplexy. Should they be situate on the arteries in the substance of the brain, rupture may result in paralysis.

Diagnosis and Prognosis.—Apoplexy must be distinguished from syncope, from hysterical coma, from alcoholic intoxication, from uræmic coma, and from the coma which sometimes follows epileptic fits, which last it is almost impossible to diagnose, except by the aid of a previous history of epileptic attacks.

Syncope is of brief duration, and is characterised by extreme pallor, coldness of the surface, sighing breathing, and great feebleness of the pulse.

In hysterical coma the sex and age of the patient should be considered.

The history of the case, and the presence of the smell of alcohol, if the alcohol be not administered by a bystander by way of treatment, will help to form a correct diagnosis in most cases. The patient also in most cases of alcoholic poisoning can be temporarily roused. Uræmic coma in most cases begins with convulsions, and there are evident and well marked signs of dropsy. The temperature also falls on the occurrence of the coma. The diagnosis from narcotic poisoning is given in a Table, p. 366.

The *Prognosis* must always be guarded, the most hopeless cases often recovering, and the most hopeful dying suddenly. A sudden fall in the temperature points to a fresh hemorrhage. The paralysis of the leg generally disappears before that of the arm.

It must be borne in mind when paralysis occurs, that the injury to the brain is on the opposite side to that of the paralysed limbs. The upper part of the face is an exception to this rule. Should recovery take place, there is generally some impairment of the functions of the mind left behind.

Treatment.—This will depend upon the nature of the case, and the condition of the patient when first seen. If active congestion be clearly made out, the prompt abstraction of blood is advisable. Should the stomach be over-loaded, as when apoplexy occurs after a meal, and vomiting be absent, an emetic to relieve the stomach may be given with caution, as the act of vomiting has a tendency to increase the congestion of the head. Purgatives in most cases are indicated, and those acting most expeditiously are to be preferred. Stimulating injections are also useful. The patient's head should be raised in bed on pillows, and ice applied to the vertex, whilst hot bottles or mustard plasters are kept to the feet and legs. The application of a large blister to the back of the neck often affords relief. The bladder must be examined from time to time, and the urine drawn off if necessary. Should the patient recover, the after-treatment must be more or less prophylactic, and measures taken to ward off a future attack. All unnecessary excitement should be avoided, the bowels kept carefully regulated, and the diet limited, no alcohol being allowed.

TABLE SHOWING POINTS OF DISTINCTION BETWEEN APOPLEXY AND NARCOTIC POISONING.

APOPLEXY.	NARCOTIC POISONING.
1. Apoplexy *may* be preceded by premonitory symptoms, giddiness, headache, nosies in the ears, and partial paralysis.	1. No premonitory symptoms, except by fortuitous combination.
2. Apoplexy chiefly attacks the old, and is very rare in young people.	2. More frequently in the young, especially of the female sex.
3. Most frequently among fat people.	3. In fat or thin people.
4. Symptoms may come on during the meal or *immediately* after.	4. An interval of from ten to thirty minutes always occurs, even in the case of opium, the commonest of narcotic poisons.
5. Symptoms commence abruptly, frequently with deep stupor.	5. Symptoms advance gradually. The *onset* of coma is *slow*.
6. Patient is with difficulty, if ever, temporarily aroused. Convulsions common. Face bloated. Pupils *dilated*.	6. Patient may be roused from the deepest lethargy if shaken or spoken to in a loud voice. Convulsions *rare* in opium-poisoning. Face seldom bloated. Pupils *contracted*. In this it closely resembles *hæmorrhage* into the *central portions of the pons*.
7. Life may be prolonged for a day or more. Apoplexy *may*, however, kill in an hour.	7. Life seldom prolonged beyond six or eight hours. The shortest time in which opium has caused death is *three* hours.
8. No response when the forehead is smartly tapped with the finger nails, or when water is injected into the ear.	8. The patient may be roused by tapping the forehead, etc.

TABLE GIVING THE DIAGNOSIS OF HÆMORRHAGE FROM EMBOLISM AND THROMBOSIS.

HÆMORRHAGE.	EMBOLISM AND THROMBOSIS.
1. Onset sudden, preceded by prodromata in old people.	1. Gradual onset, with premonitory symptoms in old people—*thrombosis.* In young persons, without prodromata —*embolism.*
2. Deep and lasting coma.	2. Coma deep, etc., if several small vessels suddenly plugged.
3. No remission of symptoms. Signs of inflammatory reaction.	3. Occasionally, remission of symptoms about third or fourth day—*embolism.*
4 Temperature lowered; then rise of some degrees above the normal, more or less lasting.	4. Sudden rise of temperature at outset, followed by a speedy and marked fall.

The following Table gives the kinds of Tumours most frequently found in the Cranium :—

GLIOMATA OR GLIO-SARCOMATA.	SCROFULOUS OR TUBERCULAR.	SYPHILITIC OR GUMMATA.
Proliferation of the cement substance of the neuroglia. Vary in size from a cherry-stone to half the brain. Often very vascular; contain cheesy matter in substance of tumour. Slow growth, seldom circumscribed.	Firm yellow nodules with vascular cortex. Size of hen's egg. Often cystic.	Irregular form, firm, pinkish-purple colour, not easily distinguished from gliomata or scrofulous tumours. Sometimes appear translucent or gelatinous; margin seldom distinctly circumscribed. More common form of tumour in the adult.

N.B.—The symptoms to which intercranial tumours give rise are sudden paroxysmal attacks of headache, vomiting occurring with or without the headache. Giddiness, mental disturbances, as loss of memory, irritability of temper, etc. Epileptiform attacks, the convulsions limited to one side, with visual or auditory aurœ are not unfrequent. *Double optic neuritis*, especially when present with the above symptoms, in the absence of lead poisoning and kidney disease, is almost decisive in favour of cerebral tumour.

Table giving Diagnosis of Tumours of the Brain.

1. Pressure on the third nerve causes—(*a*) External divergent strabismus. (*b*) Ptosis. (*c*) Dilatation of the pupil. (*d*) Diplopia, or double vision. (*e*) Want of accommodation for near objects.
2. Pressure on the fourth nerve.—Inability to roll the eye downwards and outwards.
3. Pressure on the optic tract.—Hemiopia of both eyes.
4. If growing in centre of optic tract and pressing only on anterior commissure.—Hemianopia of inner side of both eyes; *i.e.*, Heteronomous Hemianopia.
5. If occipital lobe injured on right side.—Hemiopia of the opposite side.
6. Pressure on under surface of the right crus.—Left Hemiplegia.
7. Pressure between the crura.—Paralysis of both sides, and double Ptosis.
8. In the cavity of the fourth ventricle.—Reflex action interfered with; respiratory centre affected; death from asphixia.

APHASIA.

Definition.—A symptom of a diseased condition of the brain, brought about by a variety of causes, and characterised by the loss of memory, of words, or the right use of words.

A patient recovering from an attack of apoplexy finds that, together with loss of motion on the right side, he has also lost the power of intelligent speech. Sometimes the memory of words is entirely lost, *amnesic aphasia;* or there may be an inability to perform the movements required for articulation, *ataxic aphasia.* In another form of aphasia—*aphemia*—the patient is quite speechless. But it must be borne in mind that, although hemiplegia is a frequent concomitant of aphasia, it is not invariably present. True aphasia must be distinguished from inability to speak from paralysis of the muscles of the tongue, met with in glosso-laryngeal paralysis and some other affections; and it must also be separated from aphonia or loss of voice, the patient in this affection being able to whisper without any disturbance in the idea of speech. In many cases of aphasia the patient loses the power of using the proper words to express his wants; thus, asking for a stick when he is in want of his coat or boots; sometimes a short sentence is used in reply to every question, the patient appearing conscious that his words do not convey the ideas he wishes to express. A case of well-marked aphasia with slight paralysis, consequent on the presence of a large extravasation of blood over the left frontal lobes, was seen by the writer at the Royal Dispensary, Edinburgh. The patient said "No, no," to every question asked.

Pathology.—The pathology of the disease is still somewhat obscure, but it appears to be connected with disease of the posterior portion of the third left frontal convolution or lobe of Broca, due to embolism of the left middle cerebral artery which supplies that portion of the brain. Aphasia may arise from simple *vaso-motor* disturbance of the part just mentioned, and this may account for the temporary forms of disease sometimes seen. This artery supplies blood to the corpus striatum on the same side, hence the frequent accompanying hemiplegia when any obstruction takes place in the artery, impairing nutrition, and thus preventing the performance of the healthy function of the parts. Dr Hughlings Jackson is of opinion that embolism of the artery is the immediate cause, and this may follow heart disease, the result of previous attacks of rheumatic fever. The presence of aphasia points to the fact that beyond the circle of Willis there is no anastomosis of the cerebral vessels. If only the grey matter of the anterior lobes is affected, there is loss of memory, of words, without hemiplegia, and there is no difficulty of articulation, *amnesic aphasia;* when the corpus striatum is also implicated, *ataxic aphasia*, hemiplegia present. The presence of both, or absence of either, points to the seat of the brain lesion.

SUNSTROKE.

Lat. *Solis ictus.* Fr. *Coup de soleil.* Ger. *Sonnenstich.*

DEFINITION.—*Insolation*, *Coup de soleil*, or Sunstroke, are terms applied to an affection of the nervous system due to exposure to intense heat and the sun's rays.

Sunstroke is more frequent in tropical than in temperate climates, though children have frequently been attacked in England during hot summers. Debility from disease, intemperance, and insufficiency of good food and water, are among the predisposing causes.

Symptoms.— Some cases are marked by extreme exhaustion, the vital forces being prostrated, and the patient dying in a state of syncope. For some days previous to the attack, the functions of the skin appear to be arrested, the surface of the body being dry and entirely free from perspiration. The attack generally comes on during the night or late in the evening. The effect on the nervous system is shown by the accompanying headache and vertigo—congestive apoplexy—followed by a loss of consciousness, stertorous breathing, and convulsions. Paralysis does not accompany or follow an attack of sunstroke. The pulse, frequent and full at the outset, becomes, as a fatal termination draws near, frequent and feeble. The seizure in most cases is abrupt, the premonitory symptoms being slight or wanting.

Dr Swift, in the *New York Journal of Medicine*, gives the following account of the attack:—"The patients are suddenly seized, whilst in the performance of their labours, with pain in the head, and a sense of fulness and oppression in the epigastrium, occasionally nausea and vomiting, general feeling of weakness, especially of the lower extremities, vertigo, dimness of vision, and insensibility. Surrounding objects appear of uniform colour. In a great majority of cases, this was, so far as could be ascertained, blue or purple. In one instance everything appeared red, in another green, and in another white."

The **Post-mortem** appearances, in some cases, are those of death by asphyxia; the lungs are congested, and the vessels of the brain, choroid plexus, etc., are engorged with blood. According to Morehead, the blood is always fluid. This gentleman is also of opinion that depression of the functions of the cerebro-spinal and sympathetic nervous systems is chiefly concerned in the phenomena of sunstroke.

Treatment.—The treatment will depend upon the condition of the patient. If nervous exhaustion be a prominent symptom, perfect rest is absolutely necessary, and stimulants should be gradually and carefully administered. If the surface of the body be hot, sponging with cold water will give great relief. The cold douche has been found of great service. Blisters or mustard-plasters to the neck and calves of the legs are sometimes of use. The use of stimulating enemata has been strongly recommended by Assistant-Surgeon Chapelle, of the Royal Artillery.

All depressing measures—blood-letting, emetics, and cathartics—should be avoided, unless the case present well-marked apoplectic symptoms.

DELIRIUM TREMENS.

Lat. *Delirium alcoholicum.* Fr. *Delirium tremens.* Ger. *Delirium tremens.* Syn. *Säuferwahnsinn.*

DEFINITION.—*Delirium tremens, delirium è potu,* or *delirium ebriositatis,* are the names applied to a disease induced by the abuse of spirituous liquors.

Delirium tremens is placed, in the nomenclature of diseases published by the College of Physicians, among the results of poisoning by alcohol.

Symptoms.—After a period of continuous indulgence, the patient complains of great depression both of body and mind. His face is pale, and he suffers from a sensation of chilliness; he also dreads to be alone, and is haunted by spectres. A state of active excitement now comes on, in which he gets no sleep, but talks continually in a timid, half-suspicious manner; he expresses his belief that his friends are plotting against him, and is constantly anxious to get up and escape from murder, fire, and thieves, against which he loudly vociferates. He may be sometimes found hard at work sweeping "black devils" out at the bottom of his window as fast as they come in at the top. In fact, there is no disease which presents such a variety of delusions, often of the most ludicrous nature. His skin is clammy, and his pulse weak and compressible; his muscles tremble, and even his tongue when put out trembles, or is instantly drawn into his mouth again. His conjunctivæ are red and suffused, his eyes restless, and he is constantly fidgeting with his hands and picking at the bed-clothes. His tongue and breath are foul, and his stomach irritable; but yet he frequently declares that there is nothing the matter with him. The attack generally lasts about three or four days; then, having worn itself out, ends in sleep, but sometimes the exhaustion proves fatal. An attack of delirium tremens may occur in habitual drunkards during the course of acute diseases—pneumonia, bronchitis, etc.—although alcohol had not been taken for some weeks previous. In the majority of cases these end fatally; but, as a rule, simple uncomplicated delirium tremens ends favourably.

Treatment.—The treatment consists in stopping the use of all alcoholic drinks, in clearing the bowels out with a saline purge, and then attempts may be made to procure sleep. For this purpose opium has been used largely, but it is now generally held that the hydrate of chloral is preferable, and may be combined with bromide of potassium. Cold to the head, or the use of the cold shower-bath, has often been attended with beneficial effects. Dr Jones, of Jersey, recommended the administration of digitalis. He gave half an ounce of the tincture for a dose, to be followed in four hours by the same quantity, if necessary. The bromide of potassium alone, given in large doses, is often followed by marked improvement in the condition of the patient. The patient should be well fed, any objection on his part to food should be met with kind but determined firmness.

The general treatment will consist in enforcing the most absolute quietude, and by allowing a light and nutritious diet.

INSANITY.

To attempt to define insanity—even if it were possible—in so short a work as the present, would be futile. I shall therefore adopt the following classification, which, if it be not absolutely correct, has this one advantage—simplicity:—

(*a*) Idiocy, Cretinism, and Imbecility.
(*b*) Dementia.
(*c*) Monomania.
(*d*) Melancholia.
(*e*) Acute mania.

(*a*) IDIOCY.—Idiocy is congenital, and was defined by Esquirol thus: "Idiocy is not a disease, but a condition in which the intellectual faculties are never manifested, or have never been developed sufficiently to enable the idiot to acquire such an amount of knowledge as persons of his own age, and placed in similar circumstances with himself, are capable of receiving. Idiocy commences with life, or at an age which precedes the development of the intellectual and affective faculties, which are from the first what they are doomed to be during the whole period of existence."

Cretinism differs from idiocy in being endemic; it is also more curable, or at least more susceptible of improvement, than the latter. In the idiot the malady is congenital; the cretin, on the other hand, may to all appearances for a time be free from disease. "Every cretin is an idiot, but every idiot is not a cretin; idiocy is the more comprehensive term, cretinism is a special kind of it." The enlarged thyroid gland, high-arched palate, and brown or yellow colour of the skin, are characteristic of the cretin. Local causes seem to be at work in the production of cretinism, but what the exact nature of these causes is has not been definitely settled. It has been attributed to miasma, to overcrowding in low-lying, badly-ventilated houses, and to ill-assorted marriages. Smallness of the brain, premature ossification of the cranium, and want of symmetry in the brain, have also been mentioned among the causes of cretinism. The idiot is usually cunning, mischievous, and dirty in his habits.

Imbecility is a minor form of idiocy, and may or may not be congenital.

(*b*) DEMENTIA consists in a failure of the mental powers coming on during life. The mind becomes weak and the ideas confused. "A man," says Esquirol, "in a state of dementia, is deprived of advantages which he formerly enjoyed. He was a rich man, who has become poor. The idiot, on the contrary, has always been in a state of want and misery." In this state there is always more or less incoherence, and maniacal paroxysms are not unfrequent. Among the causes of dementia may be mentioned fright, a previous attack of acute mania, or it may follow in the train of general paralysis.

(*c*) MONOMANIA is a form of mental exaltation characterised by the existence of one particular idea, which becomes the ruling passion of the individual. "The monomaniac lives *without* himself, and differs from others in the excess of his emotions. The physiognomy of the monomaniac is animated, changeful, pleasant: the eyes are lively and brilliant. The monomaniac is gay, petulant, rash, and audacious." Beyond the one engrossing idea, the monomaniac may show no obvious disorder of the mind.

(*d*) MELANCHOLIA.—A form of mental derangement generally classed under the head of *Delusional Insanity.* The mind becomes depressed with mournful forebodings, and the sufferer becomes careless as to his personal appearance, and in everything that happens around him he finds new sources of pain. Religious melancholia among women is not uncommon. Suicide is not unfrequently attempted by these unfortunates.

(*e*) MANIA.—A derangement of the mental faculties, attended with sudden outbursts of ungovernable fury.

Diagnosis.—In *delirium tremens* there is a peculiar nervous tremor not noticed in mania. The illusions and hallucinations in *delirium tremens* are generally of a fearful nature. The victim is either pursued by devils, by robbers, or by his own friends, or he has an urgent desire to do something more or less connected with his daily occupation. The man, if a wine merchant, will tell you that he must be engaged bottling wine, and all your efforts to dissuade him only increases his anxiety to be at work. The physical condition also differs from that of acute mania:—the skin is cooler and more clammy, the pulse is weaker, and the tongue whiter and more tremulous.

In *Meningitis* the tendency to muscular exertion, so common in acute mania, is absent, or only exercised for a very limited period. The maniac will pace up and down his room for hours and days. In meningitis also the pulse is full and bounding, the eyes bloodshot, the pupil contracted and unaffected by light, and the skin hot and dry. There is also excessive cephalalgia, rigors, and convulsions, followed by death.

In *Mania* there is a characteristic expression of the face, which need be only once seen to be ever remembered. There is frequently loss of sleep, and a man suffering from mania will keep constantly talking, night and day, for a week or more. This prolonged sleeplessness cannot be maintained by an impostor, who generally finds himself overcome by the demand for sleep. The maniac cannot sustain any prolonged conversation; in fact, in most cases conversation is impossible.

In *Febrile Delirium* there is a history of a particular fever. The delirium is low and muttering, and the fits of excitement only temporary; besides, the patient can in most cases be recalled by a sharp question to give a rational account of himself.

Puerperal Mania is much allied to ordinary mania. It may partake of the character of delirium and furious excitement, or a low form of melancholia. The former is the more frequent. During the attack it is singular to note that the patient is conscious that she is the subject of delusions. Many cases, according to Dr Gooch, present symptoms not unlike those of *delirium tremens*, and he also remarks that "nervous irritation is very common after delivery, more especially among fashionable ladies, and this may exist in any degree between mere peevishness and downright madness." In many cases there is a great tendency to suicide, or to injure the child.

The following case is given in the graphic language of Dr Gooch:—"Can you prevent puerperal mania? A lady, a few days after labour, became maniacal. The symptoms subsided in about five weeks, and she got well. She again became pregnant, and I attended her, but was ignorant of her former malady, until the nurse informed me of it in the progress of the labour; and if hereditary predisposition have anything to do with this disease, this must have been an emphatic instance, for nearly all her relatives were mad, or had died mad; and, to keep up the breed, this lady had married a gentleman whose family were equally mad. After her first labour, her friends, thinking it a time for merriment and rejoicing, were footing it about the house, which resembled a rabbit-warren. I determined it should this time be otherwise, and succeeded in keeping the house quiet. She was delivered easily, and did well for the first ten days, when, as the devil would have it, a fire broke out near the house in which she lived. The attendants very properly kept her ignorant of the circumstance; but in the evening she saw some sparks flying about. Almost immediately after this I happened to call, thinking the family would be in a state of alarm. She looked and talked rather oddly, and the paroxysm was evidently coming on. I slept in the house that night, and at two o'clock was called up. On entering her bedroom she said, 'Who is there?' I merely answered, 'Dr Gooch.' She replied, 'Sit down; now look at my forehead; do you see anything?' 'No, ma'am.' 'Look again.' 'I see nothing there.' Then, clasping her hands, with a whining, methodistical tone, she exclaimed, 'Then I was presumptuous; I am deceived. I thought a glorious light issued from my temples, and that I was the Virgin Mary.'" This lady ultimately got quite well, "without seeming to have been cured by anything that was done for her."

Treatment.—The best treatment for puerperal mania is perfect quiet, regulation of the bowels, change of air, and cheerful society. Never argue with the patients about their fancies. "I would rather," says Dr Gooch, "allow a patient to think her legs were made of straw and her body of glass than dispute either proposition."

GENERAL PARALYSIS OF THE INSANE.

Lat. *Paralysie insanorum.* Fr. *Paralysie des aliénés.*
Ger. *Allgemeine Lähmung der Wahnsinnigen.*

General paralysis of the insane, or perhaps a better term, *progressive paralysis of the insane,* or *paralytic dementia,* is a disease about which opinions vary greatly. The disease is considered by some to precede the psychical derangement, a contrary opinion being held by others. General paralysis may accompany any of the forms of mental derangement, but it is generally preceded by a melancholic stage. The disease is more common in men than in women.

Symptoms.—These are often very insiduous, for, in many cases, long before there is any marked symptom to attract attention the whole character of the patient has undergone a serious and most important change; thus he may gradually lose all sense of duty, becoming careless as to his person and dirty in his habits—what is sometimes described as "very eccentric of late." He is very loquacious, and expresses himself as possessed of great property, and boasts of the wonderful deeds that he can accomplish or has accomplished, often ruining himself or friends in the prosecution of his wild fancies and beliefs. After the above symptoms have continued for a time, the paralytic character of the disease appears; the symptom first attracting attention, and which is perhaps the first in the order of sequence, being a modification in the articulation. "This is neither stammering nor hesitation of speech. It more closely resembles the thickness of speech observable in a drunken man. It depends upon the loss of power over the co-ordinate action of the muscles of vocal articulation." If the tongue be now examined, it will be found that when it is protruded it is not inclined to one side, but that it is tremulous, and is protruded and withdrawn in a convulsive manner.

Griesinger was the first to call attention to the fact—and his statement has since been confirmed—"that this motory disorder is at the commencement not so much paralytic as convulsive in its nature." The lips have also a twitching action, even when the rest of the face is in repose. The gait then becomes unsteady, he walks stiffly, and stumbles over the slightest unevenness in the floor. Step by step the paralysis progresses, till at last the unfortunate sufferer takes to his bed, on which he may lie for some months. All the time, that is as long as he can speak distinctly, he is fond of stating his intense happiness and recounting his enormous wealth. Sometimes, especially during the earlier stages, he may suffer from terrible delusions, from maniacal paroxysms, or from epileptic fits, the latter possessing certain peculiarities. The tongue during the fit is seldom bitten, which is so commonly the case in epilepsy; and the convulsions are not so general, being limited more to one side than the other. It is also remarkable that each fit is in most cases followed by an increase in the mental derangement. The stupor which follows the epileptiform seizure of general paralysis is distinguished from that accompanying apoplexy by the absence of stertorous breathing and the puffing of the cheeks in the latter disease. The paralysed limbs are usually rigid, and not unfrequently convulsed, which is not the case in sanguineous apoplexy. During the earlier stages of the disease both psychical and motory remissions may occur, and may last for several weeks.

AGE, CAUSE, AND DURATION.—The disease seldom occurs before twenty years of age, generally about forty, and very rarely after sixty. The educated classes most frequently suffer. It is said to be caused by drinking, syphilis, excessive venery or study, and the abuse of tobacco and opium. The duration of general paralysis may vary from six months to three years or more.

The **Post-mortem** appearances of the brain show no one particular condition to which this disease may be attributed. General destruction of some of the important parts of the brain and spinal cord, the result of a chronic inflammatory process like that of Bright's disease of the kidney, may be found. The pia mater and arachnoid are thickened, and adherent to the grey matter of the brain. The vessels of the brain undergo several changes; some are calcareous, others have their coats thickened, and appear varicose. After recounting the several changes found in the brain of the insane, Wilks and Moxon remark, "We must warn you that there is nothing peculiar in their kind. *There is no one of these changes which may not be found in ordinary brains after middle age, or even younger.* The most characteristic point with regard to insanity is the universal spread of these changes. Local causes of irritation, such as tubercles and hydatids, do not of themselves produce insanity; they are rarely found associated with it. To produce insanity, the morbid changes must be widely diffused in the grey matter of the surface." M. Calmeil says that there is "some one modification of the brain whose nature we have not yet learned to appreciate." Dr Bucknill believes "that general paralysis is essentially *a disease of nutrition affecting the whole nervous system.*"

Treatment.—Eserine has been recommended by Dr Crichton Brown in general paralysis of the insane.

The general treatment of insanity will be *partly moral*, *partly hygienic*, and *partly medicinal.* In most cases firmness and strict adherence to truth are essential. The diet should be nourishing and properly cooked. Healthy exercise and constant employment for the mind form a considerable item in the treatment of the insane. For the measures of treatment to be adopted in particular cases, the student is referred to special works on the subject. A short residence in an asylum will enable him to learn more of these terrible affections than he may ever hope to obtain from the perusal of books on the subject.

A Table showing the Causes of Insanity.

First—Physical Causes.

- Intemperance.
- Epilepsy.
- Affections of the Head and Spine.
- Uterine Disorders—viz., those of
    - Menstruation.
    - Pregnancy.
    - Parturition.
    - Lactation.
- Vice and Immorality.
- Fever and Febrile Diseases.

Second—As regards Moral Causes.

- Domestic Troubles and Grief.
- Religious Anxiety and Excitement.
- Disappointed Affections.
- Fear and Fright.
- Intense Study.
- Political and other Excitement.
- Wounded Feelings.

—*From* Bucknill *and* Tuke.

EPILEPSY.

Lat. *Epilepsia.* Fr. *Epilepsie.* Ger. *Epilepsic.*
Syn. *Fallende Krankheit.*

Definition.—Epilepsy is a paroxysmal affection attended with partial or entire loss of consciousness, and convulsions of a tonic and clonic character. It has been divided into two kinds, *grand-mal* and the *petit-mal*, according to the severity of the symptoms.

Symptoms.—An epileptic fit sometimes comes on without warning, but in most cases there are premonitory symptoms which give notice of the attack. These may be either a sensation of tingling or coldness, felt first in one extremity and gradually mounting towards the head, and known as the *aura epileptica;* or the patient may suffer from delusions of the senses, which always precede the attack.

The epileptic seizure—*grand-mal*—may be thus described: Sometimes, without any warning to the bystanders, the epileptic suddenly, with a loud shriek, falls, bereft of consciousness, but strongly convulsed. The eyes are generally wide open, the pupils dilated, and the eyeballs turned up and rolling from side to nyst side. The breathing stops, and the voluntary muscles agmu are at first rigidly contracted; this condition lasting from *ten* to *thirty seconds*, and then followed by tonic and clonic convulsions, the muscles being contracted and relaxed in rapid succession. The tongue may be protruded, and in most cases is severely bitten. At first there is a white foam at the mouth, becoming tinged with blood, from the lacerated edges of the tongue. The inspirations are impeded, and are accompanied with a hissing or sighing noise, due to the spasm affecting the muscles of the larynx. The action of the heart is tumultuous. After a period more or less prolonged, the patient gradually recovers consciousness, but with a more or less scared expression of the countenance.

In the *petit-mal* a mere momentary faintness and loss of consciousness, without spasm, characterises the attack. The patients often describe the fits as "turns," "faints," or "strange sensations" coming over them. Patients may bruise their hands against the bed-posts, when the fits come on during the night, and be unconscious of the fact. The writer has the notes of a case which occurred during his residence at the City of London Lunatic Asylum, of a young man the subject of epileptic fits. This poor fellow seldom fell down, but would have the fits standing. He usually put his hand behind his right ear, and, making a peculiar noise like the running down of a clock, continued standing till the fit had passed off, when he would resume his employment. As a question of medico-legal interest, it must be remembered that post epileptiform acts, or the automatic performances of certain actions, are not uncommon. We have seen patients laboriously engaged in remaking their beds when there was no necessity for doing so. At first the fits occur at long intervals—weeks and even months may intervene, but as time goes on they occur more frequently, and may become an almost daily, and in severe cases an hourly, occurrence.

Etiology.—The causes of this disease have been divided under two heads, *centric* and *eccentric* or *centripetal;* the former depending on some disease or irritation of the brain, the latter depending on some distant irritation, such as intestinal worms, or something having reference to the genital organs, masturbation, etc. etc. It may certainly be hereditary. Epileptic fits are very common in confirmed lunatics. The disease is most common between ten and twenty years of age. The unstable condition of the nervous system in rickitic, badly nourished children, is doubtless a predisposing cause; whilst as exciting causes, fright, and undue excitement must not be forgotten.

At present we possess no pathological evidence as to the essential condition in epilepsy. Induration from exudation in some cases, and softening from fatty degeneration in others, have been shown from the researches of Van der Kolk to exist in the medulla oblongata in some epileptics. Dr Todd considered that epilepsy is due to the collection of a *materies morbi* in the blood acting on the brain and giving rise to an explosion. The cortex of the brain may be the seat of the discharging lesions. Van der Kolk considers an exalted sensibility and excitability of the medulla oblongata as essential to the development of the epileptic phenomena. Kussmaul and Terrner are of opinion that the convulsions are the result of an anæmic condition of the brain; and to explain the anæmia, spasm of the arteries and capillaries has by some been suggested. This vaso-motor theory is held by Gowers to be untenable. But this explanation—anæmic condition of the brain—plausible enough, does not help us in cases of obstruction to respiration, unless "we may fall back on the subtle ingenuity of Mr Simon, and say that stasis is the equivalent of anæmia; and perhaps, if we practically do so, and say that the cause of epilepsy is a negative state of blood-influence, due either to deficiency of its supply or to inefficiency of its quality, we shall have a simpler view, and useful as far as it leads us to cease regarding epilepsy as a strong and positive state."

Diagnosis and Prognosis.—Do not confound epilepsy with uræmic poisoning, lead poisoning, hysteroid seizures, and convulsions due to tumours of the brain. The prognosis is as a rule favourable, the patient living for many years.

Treatment.—The treatment may be divided into two parts—during the fit, and after the fit. During the fit, care must be taken that the patient does himself no harm. The clothes about the neck must be loosened, and in women it is as well to remove the stays. A piece of cork or other soft substance should be placed between the teeth so as to guard against any injury to the tongue. Cold may be applied to the head and hands, and the patient placed in a cool well-ventilated room. As soon as he recovers from the fit, the exciting cause must be sought and removed. In young men who indulge in masturbation, kind treatment, combined with moral persuasion, will be necessary. In girls, irregularities in the catamenia must be corrected. If caused by intestinal worms, means should be taken for their removal. Epilepsy is often shammed; an examination of the eyes will betray this. In true epilepsy these are generally open and incapable of receiving impressions, but in spurious epilepsy they are closed, and the pupils contract when a bright light is thrown upon them. The suggestion of the application of the actual cautery or red-hot poker will soon cause a return to consciousness in cases where the disease is simulated. Of late years the bromide of potassium has been much used in the treatment of epilepsy, with the marked effect of reducing the frequency of the attacks. It has been given in drachm doses three times a day in severe cases, but smaller doses are to be preferred. Dr Gowers, however, proposes to give two to three drachms every third or fourth morning, and gradually to increase the dose in about two or three weeks to six or eight drachms every fifth morning, and then to decrease the dose. Other drugs which have been found useful are belladonna, oxide, sulphate, valerianate, and acetate of zinc, conium, etc. These may often be beneficially combined. I have found great benefit from a combination of arsenic, cannabis Indica, bromide of potassium, and bromide of lithia. In traumatic epilepsy trephining the skull has been proposed, and the operation has been attended with success.

LARYNGISMUS STRIDULUS.

Lat. *Laryngismus Stridulus. Spasmus glottidis. Angina spastica. Clangor infantum.* Fr. *Spasme de la glotte.* Ger. *Mellarsches Asthma.* Syn. *Krampf der Glottis.*

DEFINITION.—This disease consists in spasm of the small muscles of the larynx, due in many cases to teething or some gastric irritation. The term "cock-crowing" has been given to this disease.

Laryngismus Stridulus is essentially a disease of early childhood, especially occurring during the first year of infantile life, and also during the period of cutting the teeth. It is the "Faux croup" of French writers, and must not be confounded with acute laryngitis, membraneous croup, convulsions, or foreign bodies in the larynx.

Symptoms.—The attack comes on suddenly; the child may be in its cradle, when it is heard struggling for breath. The countenance becomes purplish, the respiration impeded, and attended with a peculiar whistling sound during inspiration. The attack soon subsides, and the child, as soon as it recovers from its fright, may drop off to sleep again. The paroxysm, from the slightest cause, is likely to return. The following is the account of a case which occurred in the practice of the writer:—I was called one Saturday evening to see a fine healthy little boy about twelve months old, who was supposed to be suffering from his teeth. The child was fretful, but there did not appear to be very much the matter, except that occasionally he was attacked by an apparent spasm of the throat, which caused him to make a peculiar noise, like the crowing of a cock, when he drew in his breath. I ordered his diet to be carefully regulated, gave him a purge, and ordered small doses of the bromide of potassium with valerian. The gums were not full enough to justify lancing. About mid-day, on Sunday, I was suddenly sent for, and found the little fellow dead. It appeared that his father had taken a book to read, leaving the child playing close to him on the floor. Falling asleep over his book, he was suddenly aroused by the peculiar noise the child was making; catching him up in his arms, he rushed into the kitchen to procure a hot bath, but before the child's clothes could be removed it was dead. This, unfortunately, is not an isolated case.

Treatment.—The treatment consists in placing the child in a warm bath, and by adopting those measures found most efficacious in the convulsive attacks to which children are liable. Mustard plasters may be applied to the neck, or even leeches if suffocation appears imminent. The cause should then be sought and removed. If due to teething the gums may be lanced, if there is every prospect that the teeth are so close to the surface of the gum as to prevent it uniting by a cicatrise over the teeth.

CHOREA.

Lat. *Chorea.* Fr. *Chorée.* Ger. *Veitztanz.*

Definition.—Chorea, or *St Vitus' Dance*, is a disease characterised by irregular contractions of the voluntary muscles, especially of the face and arms, giving rise to movements which are only partially, if at all, under the direction of the will. These contractions become worse if the patient perceive that he is noticed.

Chorea is a disease found chiefly among children and young persons, especially girls about the age of puberty. During the middle ages it was attributed to "demoniacal possession;" in more modern times it has been described as "insanity of the muscles."

Symptoms.—The general health of the patient is, as a rule, more or less impaired, the bowels are confined, and the appetite is capricious or entirely lost. Anæmia is often a marked feature in the case. Following or accompanying the general impairment of the health just noticed, the peculiar convulsive movements, characteristic of chorea, present themselves. In chorea, if the patient tries to lift a cup to his lips, the main directions of movement are disturbed from the outset by contradictory movements, so that the goal is missed. The choreic movements also come on suddenly and unexpectedly, the patient without any apparent reason having a quiet grimace all to himself, or thrusting out his arms or legs unmeaningly. The child finds it impossible to restrain these movements, which increase if the attention of bystanders be drawn to them. During sleep the convulsive actions in most cases cease. The parts of the body, in the following order, are those most frequently attacked by the convulsive movements—the hands, right hand, the left arm, the face, left hand, etc., —in fact just those parts "which habitually combine in purposeful and emotional movements." Inorganic anæmic endocardial murmurs are not unfrequent during the course of chorea, and it also appears that the mitral valve in most of the fatal cases is studded with minute bead-like vegetations. The aortic valves may be similarly affected. These morbid appearances, according to Wilks, are the only constant ones found after death in chorea.

Pathology and Causes.—There are no pathological lesions peculiar to this disease; the softness of the brain and close juxtaposition of it to the interior of the skull, to which chorea has been ascribed, are the normal conditions of the brain in childhood.

By M. Sée and others chorea is considered to have some connection with rheumatism, and it cannot be doubted but that there is some obscure but intimate connection between the two diseases. In some cases it appears to be due to some cardiac lesion, irrespective of a previous rheumatic attack. Dr Dickinson is of opinion that it depends "on a widely-spread hyperæmia of the nervous centres, not due to any mechanical mischance, but produced by causes mainly of two kinds, one being the rheumatic condition, the other comprising various forms of irritation, mental and reflex, belonging especially to the nervous system." Kirkes, Hughlings Jackson, and others attribute the disease to a *shower of minute emboli* in the small vessels of the brain, which emboli have broken away from the vegetations on the valves of the heart. By one set of observers, the seat of the mischief in the brain is located in the corpora striata, by others in the adjoining convolutions. In many cases the disease appears to be a purely *functional* disorder affecting the motor centres of the spinal cord. Worms, fright, and an anæmic condition, have all been considered as causes of chorea. Children, especially girls, between ten and fifteen years of age, are the usual subjects of this disease, but it has been known to occur as late in life as eighty-four. The usual duration of the disease is from one to three months, but it may last as many years.

Treatment.—The treatment will consist in the administration of appropriate medicines to remove the anæmic state of the blood, if such a condition be present.

Constipation, when present, calls for remedial treatment. If the child be able to stand the shock, cold shower-baths may be tried. Iron, zinc, arsenic, and strychnia have all been tried with varying success. Anti-spasmodic remedies have also been given, among which valerian, cannabis indica, and camphor may be mentioned. Hygienic measures must not be forgotten. In the children's hospital in Paris great attention is paid to gymnastic exercises in the treatment of St Vitus' Dance. It is well not to allow a child affected with chorea to associate with others in the same nursery, as the faculty of imitation is most keen in childhood.

NEURALGIA.

Lat. *Neuralgia.* Fr. *Néuralgie.* Ger. *Neuralgie.*

DEFINITION.—A painful affection of the nerves of unknown pathology.

Varieties—(*a*) FACIAL OR TIC DOULOUREUX; (*b*) SCIATICA.

At present we know nothing of the changes in the nerve trunks or in the nuclei of the spinal cord which give rise to the disease in question, and our knowledge of it is limited to its effects and the marked symptom—pain—which accompanies it. The disease is, as a rule, unilateral, seldom attacking the corresponding nerves of the other side. Neuralgia has been attributed to exposure to cold, to carious teeth, or to injury or pressure on a nerve trunk. An important characteristic, especially of that form of neuralgia known as *tic-douloureux* or trifacial neuralgia, is the paroxysmal nature of the attack which led Trousseau to call it "epileptiform neuralgia." The pain is for the time intense, and if the disease is not soon cured the constitution of the unfortunate sufferer becomes more or less injured. If palpation or percussion be performed along the course of the nerve, certain "tender spots," usually at points where the nerve emerges from a bony cavity or under tense fascia, are detected. The cause of the tender spots is probably due to the liability of the nerve to pressure at the points just mentioned. Neuralgia is liable to attack any of the nerves of sensation, whether distributed to superficial or deep structures; thus we have sciatica when the sciatic nerve is affected, gastralgia (nerves of the stomach), and facial neuralgia or "tic," along the branches of the fifth cranial nerve, etc. Besides the pain which accompanies neuralgia we may have the local abolition of tactile sensibility, reflex muscular twitchings, congestion of the parts affected, and defective nutrition of the skin, accompanied with erythematous and herpetic eruptions as in herpes zoster or shingles, which is probably the result of a neurosis of some of the thoracic nerves.

Treatment.—In the treatment of neuralgia the cause, if it can be determined, must of course be removed—carious teeth, etc. Of external remedies the unguentum aconitia or the liniments of aconite and belladonna are most useful; equal parts of camphor and chloral hydrate painted over the course of the nerve will sometimes give relief. In sciatica, acupuncture is often of use. The digestive organs must be attended to before tonics, quinine, gelsemium, bromide of potassium, muriate of ammonia, etc., are administered. Galvanism may be employed, and also hypodermic injections of morphia.

MIGRAINE.

Lat. *Hemicrania.* Fr. *Migraine.* Ger. *Hemicranie.*

MEGRIM, PAROXYSMAL SICK-HEADACHE.

DEFINITION.—A peculiar form of paroxysmal headache, accompanied with certain strange defects of vision, anorexia, nausea, and vomiting. In most cases the disease is hereditary.

Migraine, megrim, or sick-headache belongs to that class of nervous affections to which, for want of a better, the term "paroxysmal neuroses" has been applied. It occurs more frequently in women than in men, and is more common from the seventh to the thirtieth year than after the latter period. Exhaustion, errors of digestion, of menstruation, affections of the teeth, etc., are attributed with its causation.

Symptoms.—An attack of megrim, as a rule, comes on suddenly, with pain of varying severity fixed to one spot in the brow or temple, thence spreading to other parts of the head, the scalp of which feels tender when touched. The pain may be either stabbing or cutting, and by some patients is often likened to the driving of a nail into the temple. The slightest movement on the part of the sufferer increases the pain; he therefore prefers to lie down in quiet in a dark room, as light also affects him. There is entire loss of appetite, and sometimes nausea and vomiting. A peculiar affection of the sight when present is said to precede the symptoms just mentioned. Owing to the supposed object seen taking a zigzagged outline, with angles like those of a fortification, Dr Airy proposed the term *teichopsia* (τειχος a city wall, ὄψις vision,) for the disease now under consideration. Among the less common symptoms of the disease we may have impaired sensation in one hand, difficulty of speech, muscular weakness, amounting in some cases to paralysis of the muscles of the eye, resulting in ptosis and strabismus. The pupils of the eye may become dilated or irregularly contracted, and I have seen complete *greyness of the hair* over the region of the frontal bone as a result of repeated attacks of migraine.

Treatment.—Hygienic and constitutional treatment during the intervals between the attacks must be used. During the attack, chloral, bromide of potassium, cannabis Indica, guarana, and last but not least in many cases a strong cup of bohea or coffee may be tried.

LOCOMOTOR ATAXY.

Lat. *Ataxia motus.* Fr. *Ataxie locomotrice.*
Ger. *Ataxie locomotrice.*

Definition.—Locomotor ataxy is a peculiar form of paralysis, due to disease of the postero-external columns of the cord, most frequently in the lumbar enlargement, and in which the power of regulating muscular movements is lost.

Locomotor ataxy begins insidiously, and its progress is very gradual; sometimes, however, the disease runs a rapid course. [The history of an acute case, the symptoms becoming developed in six weeks, is given in the *British Medical Journal*, vol. i., 1873.]

Symptoms.—The patient may first complain of pains in the limbs, like toothache, electric shocks, or "lightning pains," which he considers to be due to rheumatism or neuralgia, and for which he may be treated. He may also suffer from headache, referred to the back and forehead, and slight dimness of sight. There are usually considerable gastric troubles. The *gastric crisis* (Charcot), are paroxysms of pain of a lancinating character, shooting from the lower part of the abdomen to the epigastrium, and often accompanied with vomiting. "Girdle pains," as they are called, round the abdomen are also met with in some cases. A sense of muscular weakness gradually creeps over him, and rectal and bladder troubles are not uncommon, the latter ending in almost complete vesical paralysis. The desire for sexual intercourse well marked in some, is gradually lost in others. Formication may be felt in different parts of the body, and sufferer complains of a sensation of "pins and needles" in his arms and feet. These symptoms may continue for some time—three or four years—but still there are no marked symptoms of paralysis. Eruptions, somtimes papular, sometimes pustular, and at other times not unlike urticaria, make their appearance on the body, and are frequently apparently connected with the nerves along the course of which the pains are felt. Perforating ulcer of the great toe is sometimes met with in the course of the disease, due probably to inervation of the part. The pains increase in the limbs, and when the patient stands he can no longer detect any resistance to his foot, sometimes experiencing a sensation as if shod with cotton wool or walking on air. If he attempt to walk with his eyes closed, he immediately begins to totter and roll about in a half-drunken manner. Without looking, he is unaware of the position of his legs. The gait is peculiar. The foot is placed suddenly on the ground, the heel first, and then after an interval the toes follow with a jerk. The lower limbs are also thrown about in a most irregular way, and he appears to have no power to control their movements. Yet, with all this, he can resist any effort made to flex or extend his limbs against his will. Sometimes, as a first symptom, the patient finds that he has decreasing power of regulating the movements of his lower extremities; he suffers no pain in his limbs, but gradually increasing anæsthesia, which may become so marked that he may be pinched or even burnt with impunity. Mr Jonathan Hutchinson says that an early symptom is the difficulty the patient experiences in preventing himself from falling forward over the washhand stand in performing his morning ablutions.

Retarded Sensation is a phenomenon not uncommon during the course of ataxy. This may be shown by noting the time that elapses between a prick with a pin given to the patient and his recognition of the pain caused by the prick. Some seconds, ten or more, may elapse before the pain is felt. Sometimes also the sensation of pain is *persistent* after the removal of the cause; in others, the pain disappears whilst the stimulus is maintained. In many cases the eyesight becomes defective; there is either diplopia or a dimness of vision, so that he cannot distinguish small objects, or he may suffer from colour blindness. The pupils often undergo a change, and the condition known as the "Argyll Robertson pupil," in which light fails to induce any contraction, although the pupillary movements are active during attempts at accommodation. Myosis is far more frequently present than mydriasis. If the eyes be further examined with the ophthalmoscope, progressive grey atrophy of the retina is revealed "by chalkiness and opacity, with absence of the marginal rosiness of tint, and inability to trace the trunk vessels of the retina as they sink into the substance of the optic nerve."

Some slight symptoms of paralysis may now make their appearance, chiefly confined to the lower extremities. His utterance may become a little thick, and his speech embarrassed. At other times the symptoms follow the course we have before stated. A distinction must be made between this disease and paresis, or general paralysis of the insane, which it, in many cases, very closely resembles.

In paresis, mental symptoms are always present, and are the first in order of time; in ataxy, the motor precede the mental, and, as a rule, there is little impairment in the mental functions. The "patellar tendon reflex," or "knee reflex," is early impaired, and then lost, in locomotor ataxy.

Another affection met with in the course of ataxy is known as "Charcot's disease of the joints," in which the cartilages disappear and the articular ends of the bones become eburnated. Considerable discussion has of late taken place as to the identity of this affection with chronic rheumatoid arthritis. They are probably identical. The knee-joint, shoulder, elbow, and hip are affected in the order given. Well-marked pauses or remissions in the symptoms often occur, but recovery seldom takes place. The disease in most cases occurs between the ages of thirty and fifty, and is more frequent in men than in women.

THE FOLLOWING TABLE IS GIVEN BY DR SANKEY AS AN AID TO DIAGNOSIS.

PARESIS.	ATAXY.
1. Runs its course in a few years.	1. Is usually much slower, and may last ten or even twenty years.
2. Commences with mental symptoms.	2. Commences with pain in a distal nerve.
3. Is attended with libidinous ideas.	3. Is attended with absence of sexual feeling.
4. The motor symptoms are secondary in the order of time.	4. The motor symptoms are the primary phenomena.
5. Is only rarely complicated with pelvic difficulties.	5. Pelvic symptoms are a prominent feature.
6. There often is great violence.	6. The mental phenomena are imbecility and impaired memory.

Morbid Anatomy.—A diseased sclerosed condition of the posterior columns of the spinal cord, the posterior roots of the spinal nerves, and the roots of the optic, third, fifth, sixth, seventh, and ninth cranial nerves, is found after death. The median columns of the cord, or "columns of Goll," are, as a rule, affected. The membranes of the cord are also more or less thickened and opaque. The condition found is that of *grey degeneration* of the posterior columns of the cord and spinal nerves, and is in the form of bands, extending along considerable lengths of the tissues of the cord, and especially affecting those portions just mentioned. The change produced is probably the result of inflammation, and commences most probably in the nerve elements of the cord, and not in its coverings. "The degeneration consists of an atrophy, with disintegration of the nerve fibres, with proliferation of the connective tissue, giving to the columns a greyish-red transparent aspect."

The **Prognosis** is unfavourable; death may occur from intercranial mischief, or from inflammatory affections, etc.

Causes.—Exposure to damp, anxiety, mental exhaustion, venereal excesses, syphilis, etc.

Treatment.—Tonics, counter-irritation to the spine, and Faradisation, especially to relieve the lancinating pains, and for the same reason the sciatic nerve may be stretched. Phosphorus has been tried with some apparent success. Ergot, nitrate and phosphate of silver, and bromide of potassium have produced beneficial results when given in the early stages of the disease. If there is a syphilitic taint, treat that disease.

DISSEMINATED CEREBRO-SPINAL SCLEROSIS.

DEFINITION.—A disease of the nervous system, accompanied with a series of well-marked but varied symptoms, closely related to locomotor ataxy, paralysis agitans, and chorea.

Cerebro-spinal sclerosis is not recognised as a specific disease in the nomenclature of diseases put forth by the College of Physicians. In fact, it is only of late years that its true nature has been recognised. The sclerotic changes which are at the root of this disease are not localised in any one part of the nervous system, but are found in the *cerebrum*, *cerebellum*, *pons varolii*, and spinal cord; even the nerves do not themselves escape.

To this wide-spread distribution of the products of disease the diversity of the symptoms are to be attributed; yet, with all this, disseminated cerebro-spinal sclerosis admits of ready diagnosis, especially when the disease has reached its full development. Patients are not unfrequently found suffering from symptoms which betoken the presence of locomotor ataxy, chorea, paralysis agitans, and general paralysis of the insane, but this very complication is the proof of its own specific nature, and is easily explained when we bear in mind the morbid anatomy of the disease in question. Three forms of neural sclerosis have been recognised, depending upon the localisation of the sclerotic change: 1. Cerebral;

2. Spinal; 3. Cerebro-spinal—the last being, however, that most frequently met with in practice. Taking the cerebro-spinal form as typical, we shall describe the symptoms which characterise it, and then show in what they differ from other forms of nervous disease.

Symptoms.—The early symptoms of cerebro-spinal sclerosis are frequently very obscure—so obscure, indeed, as to evade detection for some months, or even years. Symptoms of ataxy may mask those of this disease when the sclerotic patches spread into the posterior columns of the cord. In some cases the patient complains of frequent and troublesome giddiness, everything appearing to whirl round him. If he take not hold of something he may fall. The gait in some cases is that of ataxy; in others, the incoördination chiefly affecting the muscles of the trunk—the patient cannot walk straight as in ataxy, but darts from side to side; in a third class, the foot appears to cling to the ground and the spastic walk, combined with tremor of the whole body, is present. The "petella reflex" is generally exaggerated, and the ankle clonus frequently present. There is also some dimness of vision, which does not, as a rule, result in complete blindness, as is the not unfrequent occurrence in ataxy. Flashes of light often precede the amblyopia. He states that there is a peculiar tremor—"*volitional tremor*"—in his body and limbs which he cannot account for, but which is absent during repose. These tremors probably point to implication of the medulla and pons. It must be remembered, however, that tremors from some unexplained cause may be absent, and that they also disappear when the limbs become permanently contracted, and also during the last stages of the disease when the strength of the patient becomes wasted. His eyes are also noticed to be constantly rolling—*nystagmus*—from side to side, which renders ophthalmic examination of them almost impossible. The "Argyll Robertson pupil" is rare, but the pupils are often irregular and in some cases "*pin-pointed.*"

In a second class of cases the early symptoms are those which betoken general weakness and impairment of muscular power. Sensation in the limbs is not altered, and there are no signs of any wasting of the muscles. The bladder and rectum give no trouble, as in some other forms of spinal disease. Attempts at recovery in these cases are not rare, the patient feeling better and hopeful of a return to his former health.

In a third class of cases the disease sets in suddenly, all the most pronounced symptoms appearing in a few days. Obscure and severe pain in the bowels, bladder, and rectum, accompanied with vomiting, is not unfrequently found among the premonitory symptoms, the patient at the same time suffering from much depression of spirits. The foregoing are the three most marked groups under which the early symptoms of the disease may be best arranged. Other symptoms then make their appearance as the disease progresses. A peculiar difficulty in the enunciation of words is well marked in nearly all the cases of disseminated cerebro-spinal sclerosis. The patient's speech is "thick," sometimes unintelligible, and the words are uttered in a slow, drawling manner—*scanning speech*—but though hesitating he does not stammer. The difficulty of speech closely resembles that of general paralysis of the insane, especially if in cerebro-spinal sclerosis the convulsive contraction of the lips characteristic of the former disease be present. The lips quiver as if the patient were cold, or like the quivering seen in some persons during fits of intense anger. The tongue may be tremulous when protruded, but it is not atrophied as it sometimes is in labio-glosso-laryngeal or bulbar paralysis. Mental derangements, sometimes assuming the form of melancholia, at other times mania with delusions, are not unfrequently present. As the fatal termination approaches difficulty in swallowing may be present, and the expression of the face becomes more and more meaningless, and stolid. Before this stage is reached a tetanic-like rigidity of the muscles of the lower extremities comes on, most marked when the patient tries to walk. The rigidity of the limbs is generally preceded by slight paralysis, one limb being first attacked. This paralysis, when present with well-marked symptoms of locomotor ataxy, is diagnostic, especially in the earlier stages, of cerebro-spinal sclerosis. In this combination we see the presence of the elementary forms of two diseases, progressive locomotor ataxy and cerebro-spinal disseminated sclerosis.

Etiology.—The causes of this disease are obscure.

Pathology and Morbid Anatomy.—Hypertrophy of the neuroglia and atrophy of the nerve cells, the sclerosed patches appearing as nodules in different parts of the brain and spinal cord.

Prognosis.—Unfavourable. The disease may continue five to ten years, and even longer.

Treatment.—Purely experimental. Galvanism to the brain and spinal cord. Belladonna, ergot, arsenic, phosphorus, etc.

GLOSSO-LABIO-LARYNGEAL PARALYSIS.

Syn. *Bulbar Paralysis.*

DEFINITION.—An obscure affection of the nervous system, resulting in "progressive muscular paralysis of the tongue, the veil of the palate and the lips," accompanied with difficult articulation and deglutition, ending in suffocation or inanition.

This disease was first clearly described by Duchenne, but was recognised by Trousseau some years before the description of it given by Duchenne. Glosso-labio-laryngeal paralysis may follow syphilis, rheumatism, or great mental anxiety. It must not be confounded with glossoplegia, facial paralysis, general paralysis of the insane, or progressive muscular atrophy attacking the tongue. The first is either preceded by, or accompanied with, cerebral mischief; in the second, the paralysis is not confined to the lips, is, as a rule, unilateral, and affects the muscles of the face more or less generally, and there is no difficulty in swallowing. General paralysis of the insane is, in its early stages, a convulsive disease, and is attended with some amount of mental derangement, and, lastly, progressive muscular atrophy seldom begins in the tongue, but should it do so, other muscles are, as a rule, affected.

Symptoms.—The early symptoms are obscure; the one which is, perhaps, first noticed by the patient is a difficulty of articulation. The tongue feels heavy and its movements are impeded. Gradually the movements of the organ become more embarrassed, and the tip of the tongue can only be pressed against the roof of the mouth by an effort on the part of the patient. Certain letters, lingual or dental consonants, are not clearly pronounced. Then follow difficulty of deglutition and threatened suffocation during the act of swallowing, fluids being ejected through the nostrils. The saliva collects in the mouth or dribbles away, as the effort to swallow it is attended with so much discomfort. His food collects between the teeth and cheeks, owing to the useless condition of the tongue, and his state is now most deplorable; the lips are wide open, the teeth exposed, viscid saliva flows from the mouth, any attempt to swallow it resulting in a suffocative attack. To catch the saliva the patient carries constantly a handkerchief up to his mouth. He gradually becomes worse and takes to his bed, lying on his side to allow the saliva to flow from his mouth, and he ultimately dies from starvation, apnœa, or arrest of the heart's action from implication of the vagus. As a rule, the mind is ununaffected, the patient being aware to the last of the hopelessness of his condition. In the acute form of the disease there is sudden paralysis of the tongue without loss of consciousnesss in some cases, although in some instances the patient may fall with a sudden cry, followed by death in a few hours or days.

Pathology and Morbid Anatomy.—The disease appears to be due to atrophy of the roots of the hypoglossal, facial, spinal accessory, and vagus, and of the motor nerve cells, from which these nerves arise. Atrophy of the hypoglossal accounts for the paralysis of the tongue; of the facial, for the condition of the lips and soft palate; of the spinal accessory, for the paralysis of the larynx and loss of voice; and, lastly, the feebleness of the heart's action and of respiration, to the vagus. The muscles affected are pale, and show signs of granular degeneration, and are, as a rule, diminished in bulk. An acute form of the disease, due probably to inflammation, embolism, or hæmorrhage in the parts affected in the chronic variety, has been described.

Prognosis and Treatment.—The prognosis is unfavourable, and the treatment, at present, unavailing.

MENIÈRE'S DISEASE—AUDITORY VERTIGO.

DEFINITION.—A disease characterised by sudden attacks of giddiness, preceded by noises in the ear, the patient frequently falling either forwards or to one side. These symptoms appear to be connected with some affection of the semicircular canals and cochlea of the ear.

Menière disease or auditory vertigo appears to be due to disease of the semi-circular canal and other parts of the internal ear. The affection is marked by deafness, tinnitus aurium, and vertigo. In some cases there may be loss of consciousness, but this condition is of rare occurrence. Frequently there is nausea, vomiting, and faintness, the patient becoming pale and his skin cold and clammy.

As regards treatment, the ears should be examined for wax or polypi pressing on the membrana tympani. Tonics may be given, and the general health, if possible, improved.

PROGRESSIVE MUSCULAR ATROPHY—WASTING PALSY.

Lat. *Atrophia musculorum ingravescens.* Fr. *Atrophie musculaire progressive.* Ger. *Progressive Muskelatrophie.*

Definition.—A chronic disease of the nervous system, characterised by progressive atrophy and wasting of the muscles, chiefly those of the hands and then of other muscles, and supposed to be the result of inflammation in the anterior cornua of the cord, the cells of which subsequently atrophy.

Pathology.—To some extent that of infantile spinal paralysis, the anterior cornua of the cord being the parts affected. The muscular atrophy is, as a rule, considerably advanced before the nerves show distinctly the presence of marked disease.

Symptoms.—The disease comes on slowly without any marked constitutional disturbance between the ages of thirty and fifty, the first symptom noticed by the patient being a wasting and paralysis of some muscle or group of muscles, chiefly those of the hand or ball of the thumb. The fingers give the hand a *claw-like* appearance, *main en griffe*, due to the wasting of the interossei muscles and binding of the fingers. From the part first attacked it extends to other parts usually symmetrically. It may begin in the muscles of the abdomen, or, in fact, in any muscle or group of muscles. Progressive muscular atrophy differs from acute spinal paralysis, in that the Faradic contractility of the affected muscles is unimpaired, unless voluntary power is entirely lost. In the former, also, there are well-marked fibrillary twitching, like worms making their way along the muscles under the skin; and, lastly, the paralysis of the muscles does not precede the atrophy. Common to the two diseases, we have decrease of temperature in the affected muscles, and absence of spinal neuralgia or tendency to bed sores. The bladder and rectum are not affected. Do not confound lead palsy with this disease. In the former the early loss of electrical contractility, slow appearance of the atrophy limited to the muscles first attacked, and the *blue line* round the penis is enough to point out the nature of the disease.

Etiology.—The result of unknown conditions, affecting chiefly adult males. May be hereditary in children. Death may result from the disease spreading to the respiratory muscles, or from some intercurrent affection.

Treatment.—Expectant. Faradism carefully applied in turn to the affected muscles.

PSEUDO-HYPERTROPHIC PARALYSIS.

DEFINITION.—A chronic disease occurring most frequently in children, and consisting in the excessive growth of the muscular connective tissue and atrophy of the sarcous elements. Groups of muscles may be thus attacked. The cause of this disease is probably due to some changes in the central nervous system, although no lesions of the nerve centres or nerves have as yet been detected.

Pseudo-hypertrophic paralysis frequently occurs in children. There is slowly increasing paralysis, and concurrently with the advancing paralysis, there is enlargement of the affected muscles, usually those of the legs and back. The disease may be divided into three stages, the last ending in complete helplessness on the part of the little patient, generally a boy. In the early stages the muscles retain their electro-contractility, but this gradually dies out as the disease progresses. As it is in the early stage when, according to Duchenne, Faradism may arrest the progress of the disease, its early diagnosis becomes of importance. The first symptom, then, is a gradual weakness of the muscles of the lower limbs, and as a result of this, the child stands with his legs apart, and his shoulders braced up, and his back arched as if to steady himself. In walking, the legs are set apart, the advancing leg being lifted needlessly high, and there is an apparent effort to balance the body. During the second stage, if the child is placed on the floor in a sitting posture, in its efforts to stand it first begins by going on all fours, and can only gain the erect posture by raising himself with the aid of a chair, table, or bed. A casual glance at a child in the early stages of the disease might lead one to remark on its fine muscular development. Sensation and control over the bladder and rectum are not lost, pointing to a healthy condition of the cord.

Treatment.—Faradism in the early stages as soon as the disease is suspected.

INFANTILE SPINAL PARALYSIS—ACUTE ANTERIOR POLIOMYELITIS.

Lat. *Paralysis infantilis.* Fr. *Paralysie de l'enfance.*
Ger. *Paralysis infantilis.*

DEFINITION.—A disease of young children, at first marked by acute symptoms, accompanied with fever, and ending in atrophy and permanent paralysis of certain muscles or groups of muscles. The cause of the disease is obscure. Dentition, measles, or other infantile diseases, appear to stand in some relation to its existence.

Infantile spinal paralysis may occur in children up to ten years of age, and appears to result from acute pigmentary degeneration in the anterior cornua of the spinal cord, followed by atrophy and disappearance of the large cells of that portion of the cord, and chiefly found in the lumbar or cervical enlargements. The disease, in its first stage, is probably inflammatory, the inflammatory action beginning, according to Charcot, in the nerve cells, thence extending to the neuroglia. There is some amount of sclerosis and ultimate shrinking of the parts attacked. The muscles rapidly shrink, and may ultimately undergo fatty degeneration.

Symptoms.—Sudden fever, sometimes attended with convulsions, but in some cases there may be absence of the fever. The duration of the feverish attack varies, the paralytic affection following closely upon it. The electro-contractility of the muscles is rapidly diminished and soon entirely lost—a most important symptom of this disease. The faradic and galvanic excitability of the nerves is soon and permanently lost, but this is not the case with the muscles, for although the muscles show a marked diminution in their response to both forms of current, the Faradic excitability is gradually and permanently lost, whilst the galvanic excitability about the *second* week begins to rise and soon increases above the normal. This constitutes the *reaction of degeneration*, generally lasting in this disease for six or eight weeks, after which the paralysed muscles gradually refuse to contract to the strongest galvanic current. There is also loss of sensation and reflex contractility, yet there is entire control of the bladder and rectum and no tendency to bed sores. The muscles may slowly regain their power, or they may remain permanently paralysed and atrophied, a very marked fall in the temperature of the affected muscles being most characteristic of this disease. The duration of the infantile paralysis may vary from a few days to several months, but improvement after ten months is somewhat rare, some amount of deformity of the limbs in most cases existing permanently.

Treatment.—Treat expectantly during early stage, and use remedies to reduce the fever. Rest and quiet for the little patient are indispensable. When the inflammatory stage has been passed try Faradism very cautiously, and give tonics, iron, etc.

SCRIVENER'S PALSY.

Lat. *Notariorum spasmus.* Fr. *Crampe des écrivains.*
Ger. *Schreibekrampf.*

DEFINITION.—A form of paralysis resulting in most cases from an over-use of certain muscles.

This disease occurs among lawyers, clerks, and others engaged continually in writing. The disease commences with a sense of weariness in the muscles of the hands and gradual loss of power, so that the hand becomes useless.

Treatment.—Treat by absolute rest, shampooing, and Faradism.

MERCURIAL TREMOR.

Lat. *Tremor ex hydrargyro.* Fr. *Tremblement mercuriel.* Ger. *Quecksilberzittern.*

Definition.—A peculiar form of muscular tremor due to the poisonous effects of mercury, and not characterised by any constant lesion. Common in looking-glass makers, gilders, etc.

Symptoms.—At first trembling of the hands and arms, slowly coming on, and then extending to other parts of the body. The joints may become painful, and the patient is at last obliged to desist from work from his inability to perform the more delicate operations of his craft. He may not be able to dress or even feed himself. The tremors at first are arrested when the patient lies down or is at rest, but as the disease advances the tremors do not cease even in the recumbent posture. The muscles of the eyes do not share in the convulsive movements, and sensation is not lost. In most cases the general health is affected, and the patient has suffered from one or more attacks of salivation.

Treatment.—Remove the cause. Iodide of potassium, etc.

SPASTIC PARALYSIS, LATERAL SPINAL SCLEROSIS.—"ERB'S PALSY."

Definition.—A disease of the lateral white columns of the spinal cord, in which there is hypertrophy of the neuroglia and atrophy of the nerve cells, accompanied with rigid contraction of the muscles. Most frequent in men between forty and fifty years of age.

The disease begins slowly with a sense of weakness and fatigue in the lower extremities, sometimes accompanied with pains in the back and limbs. Spastic paralysis has been divided into three stages. During the first, which may last some years, the patient is able to walk about by the aid of sticks. It is during this stage that the walk peculiar to the disease is seen. The patient moves forwards by the aid of two sticks, resting first on one and then on the other, lifting the pelvis and bringing his legs forwards with evident difficulty. The knees have a tendency to interlock, and the toes are scraped along the ground. In some cases the walk is a peculiar hop in which the whole body takes part. During the second and third stages he is confined to bed, his limbs having become rigid and extended. In the latter stage he is troubled with bed sores, bladder troubles, and frequent attacks of pneumonia and bronchitis, during one of which he dies. The tendon reflexes are increased; the knee reflex, lost or impaired in ataxy, is here present. The muscles do not atrophy, and during the early stages there is no derangement of sensibility, nor of the bladder and rectum.

To aid the diagnosis between primary lateral sclerosis, a very rare form of disease, and secondary sclerosis, the following points must be borne in mind. In the primary affection the weakness and the rigidity of the muscles are contemporaneous, the rigidity being more marked than the paralysis. In the secondary form the paralysis precedes for some time the spasticity of the muscles. The bladder is not affected, nor are sensory disturbances, *i.e.*, pains, girdle sensations, etc., present in the primary as they are in the secondary. Bed sores sometimes present in the secondary disorder are absent in the primary.

Treatment.—The same as for posterior sclerosis of the cord.

[TABLE

TABLE GIVING THE DIFFERENTIAL DIAGNOSIS OF THE FOLLOWING DISEASES:—

DISSEMINATED CEREBRO-SPINAL SCLEROSIS.	PARALYSIS AGITANS.	CHOREA.	LOCOMOTOR ATAXIA.	GENERAL PARALYSIS OF THE INSANE.
1. Tremors only manifest themselves on the occasion of intentional movements of some extent; they cease to exist when the muscles are abandoned to complete repose.	1. The Tremor exists as well when the members are in a state of repose as when they are set in motion by the will. The head takes no part in the shaking of the body.	1. The movements show themselves suddenly and unexpectedly when the limbs are in a perfect state of rest.	1. In purposed acts the gesticulations of chorea and the jerks of multilocular sclerosis are present. Tremor or rhythmical jerks absent.	1. Never met with in the sane. The tongue is first affected, is tremulous, and the speech hesitating.
2. The main direction of motion, as in lifting a cup to the mouth, persists in spite of the obstacles caused by the jerks or tremor, which increase as the hand nears its goal.	2. The jerks are not so great, are more rapid and regular than in disseminated cerebro-spinal sclerosis.	2. The main direction of motion is disturbed from the outset by contradictory movements which cause the goal to be missed.	2. In attempting to take up any object a sudden clutch is made, the hand being darted suddenly forward. Closing the eyes increases the incoördination of the muscles.	2. The motory disorder is at first more convulsive than paralytic. Objects are suddenly grasped, and then let fall.

3. More common between twenty-five and thirty years.	3. After forty as a rule.	3. Children.	3. Twenty to forty-five.	3. Never before twenty. More frequent in men.
4. The mode of progression may resemble that of ataxy and spastic paralysis.	4. Difficulty in maintaining the equilibrium of the body whilst walking. May run backwards or forwards.	4. Patient walks straight gesticulating all the while.	4. Want of coördination in lower limbs, hence he cannot walk unless eyes fixed on legs.	4. Gait of a drunken man, trips over slight objects.
5. Sclerosed or indurated patches scattered throughout the nervous system, hence simulating the diseases in this Table.	5. Absence of a pathological lesion, hence a neurosis.	5. A neurosis.	5. Sclerosis of the posterior columns of the cord.	5. Uncertain pathological lesions.

N.B.—In cerebellar tumours the optic neuritis, vomiting, and paroxysmal headache, these most prominent symptoms are wanting in cerebro-spinal sclerosis.

HYSTERIA.

Lat. *Hysteria.* Fr. *Hystéric.* Ger. *Hysteric.*

Hysteria, with few exceptions, is a disease peculiar to females, especially about the age of puberty, and then generally has some connection with the development of the reproductive organs. Some have considered it to be due to irritation of the ovaries, probably the result of a sub-acute inflammation of those organs; others to the rapid development of the ovaries. Hence the name hysteria (*υστερα*, the womb,) is inapplicable. The forms under which this disease makes its appearance are legion.

Symptoms.—As it occurs in young women, a hysterical fit comes on with immoderate bursts of sobbing or of laughter; or they may alternate, the change from one to the other being abrupt. A choking sensation, as if due to the presence of a foreign body, is felt in the throat, and is known as the *globus hystericus*, being most probably due to the spasm of the muscles of the pharynx, or, as some think, produced by the collection of flatus rising from the stomach through the œsophagus. During the fit the woman does not entirely lose consciousness, and may all the time be aware of what is going on around her, though she has not the power to speak. The tongue is not bitten, nor does she otherwise injure herself. As soon as the convulsions cease, the consciousness returns. An abundant discharge of pale-coloured urine of low specific gravity generally follows the fit. So much for a hysterical fit. The disease, in by far the greater majority of cases, has certain affections with which it is invariably associated; among these may be mentioned spurious disease of the hip, neuralgia, functional affections of the heart, hyperæsthesia of various parts of the body, hysterical coma, etc.

The **Diagnosis** of the real disease from the feigned is not always very easy, and the physician will have to be a man of many resources. By care he may detect that, if the attention of the patient be momentarily distracted, a joint may be moved which a minute before was immovable. It is impossible to lay down rules for the detection of a disease that defies all rules.

Treatment.—The treatment during the fit will consist in sprinkling the face with cold water, and in the administration of some one or other of the anti-spasmodics, the chief of which are valerian and asafœtida, together with camphor water. Tonics, change of air, and sea-bathing will assist in averting the tendency to future attacks. The state of the uterine functions must be inquired into, and any derangement corrected.

CATALEPSY.

Lat. *Catalepsis.* Fr. *Catalepsie.* Ger. *Catalepsie.*

This affection is doubtless but one of the forms under which hysteria presents itself to our notice. It may be best considered as hysterical coma, to which is superadded a peculiar rigidity of the voluntary muscles, the limbs remaining fixed in the position in which they may be placed by another. It is a paroxysmal affection, well-marked cases being extremely rare.

The treatment will depend upon the nature of the affections with which it may be allied.

TETANUS.

Lat. *Tetanus.* Fr. *Tétanos.* Ger. *Tetanus.*
Syn. *Starrkramph.*

Tetanus is a disease consisting in powerful tonic contractions of some of the muscles of the body without alternations of relaxation. When the muscles of the jaw are thus permanently contracted, the disease is known as *trismus*, or lock-jaw. Deglutition then becomes almost impossible. The body is sometimes curved forwards, *emprosthotonos;* sometimes backwards, *opisthotonos;* and sometimes to one side, *pleurosthotonos.* Tetanus may be either idiopathic or traumatic; the latter is the most frequent.

Symptoms.—Pain and stiffness of the muscles of the neck, and also of the jaws, come on suddenly, sometimes following an apparently insignificant wound. Difficulty in opening the mouth and moving the head is now experienced by the patient. The stiffness gradually increases, and the muscular spasm becomes so great that the body is bent in one of the ways mentioned above. The spasms are sometimes paroxysmal, but between the spasms the patient's muscles do not become relaxed, but he lies probably on his back quite stiff. The jaws can only be separated with difficulty, and then only to a most limited extent. The face wears a painful expression, the mouth is full of bloody saliva which he cannot swallow, the breathing is rapid, the inspirations and expirations being shortened, and he may complain of soreness in the muscles, due to the powerful contractions to which they are subjected. The pulse is rapid and feeble, the skin bathed in profuse sweat, the urine scanty, and the bowels confined, but there is no relaxation of the sphincters. The patient complains of want of sleep. The temperature varies from 101° to 103° F., but has been known to rise to 112° F. prior to death. According to Althaus, the temperature of the muscles before a convulsive attack is lower than that of the rectum, but after the fit it is higher in the muscles. The patient may die by asphyxia. Besides the above forms, we have *trismus infantum* or *neonatorum*, affecting children soon after birth, and generally due to some mischief commencing about the umbilicus; hysterical tetanus occurring among hysterical women, and tetanus due to poisoning by strychnia. The disease may be partial as in *trismus*, or general as in *opisthotonos.*

Morbid Anatomy and Pathology.—To the naked eye the cord in many cases presents little, if any, variation from the healthy standard. The vessels are, however, often congested, especially when death is the result of asphyxia, but this congestion may be entirely absent. The microscope does not aid us much in tetanus, and the changes in the cord when found are by no means characteristic of this disease. Wilks thinks that "the granular states described by Dr Clarke and others are widely open to the suspicion that they are of *post-mortem* origin." The muscles may, from the violence of the spasms, be found lacerated; the heart sometimes contracted, sometimes flabby; the brain healthy and the lungs engorged with blood, this condition depending much on the immediate cause of death. When arising from a wound, it should be examined for foreign bodies. The pathology of tetanus is obscure; it may have some relation to malaria, especially if an open wound be present. Sir T. Watson and Dr Richardson have suggested the presence in the blood of some endopathic poison not unlike strychnia in its effects.

Treatment.—Chloroform, to allay the spasms; purgatives, calomel, etc.; bleeding in some cases; frictions down the spine; hot air baths; belladonna, woorara, opium; cold shower baths; in fact, nearly every conceivable method of treatment has been tried with varying success.

THE FOLLOWING TABLE WILL ASSIST IN FORMING A DIAGNOSIS OF DEATH—THE RESULT OF DISEASE—OR BY STRYCHNIA.

TETANUS FROM EXPOSURE TO COLD OR WET, OR THE RESULT OF A WOUND.	TETANUS FROM STRYCHNIA.	HYSTERIA.	EPILEPSY.	TETANUS OCCURRING DURING THE ACTION OF OTHER POISONS.
1. Presence of wound. Symptoms have no connection with any liquid or solid swallowed.	1. Some solid or liquid taken within a short time of the commencement of symptoms. Not connected with any peculiarity of constitution.	1. Connected with a peculiar constitution. Rare in males.	1. Previous history of epilepsy.	1. Presence of other symptoms of poisoning peculiar to certain poisons.
2. Gradual accession and progress of the symptoms; difficulty in swallowing; stiffness of the jaws, neck, trunk, legs, and arms. Hands not generally affected.	2. Symptoms sudden and violent. All the muscles are affected at one and the same time. Arms affected, and the hands clenched at same time as the body and	2. Presence of known signs of hysteria.	2. Presence of the *aura epileptica.* Tongue bitten; insensibility lasting for some time.	*Obs.*—Arsenic, antimony, and other irritant

	legs. Jaw not affected or fixed, only during efforts to swallow.			poisons may sometimes cause tetanic spasms; but other symptoms are present which point to the nature of the poison.
3. Curving of the spine forwards not primarily present; generally comes on after some days of previous illness.	3. Opisthotonos, an early symptom generally appearing in a few minutes from the commencement of the illness.	3. Spasms frequently convulsive, alternating with stiffness of the muscles. Loss of consciousness.	3. Alternate contraction and relaxation of the muscles.	
4. Symptoms may undergo abatement, but there is no perfect intermission.	4. Intervals of complete intermission.	....	....	....
5. Death after the lapse of several hours or days. Direct injury to spinal cord may give rise to tetanus and death in a few hours. Recovery slow.	5. Death usually occurs in three hours, or even less than a quarter of an hour. Recovery in a few hours.	5. Never fatal. Recovery very rapid.	5. Is seldom fatal during the first attack.	....

PARALYSIS.

Lat. *Paralysis.* Fr. *Paralysie.* Ger. *Paralyse.*

Taken in the ordinary acceptation, the term paralysis is used to imply a loss of power in the voluntary muscles. This term is, however, also applied to denote a diminution or loss of general or special sensibility. When both motion and sensibility are lost, *perfect paralysis* is said to exist; when only one, *imperfect paralysis.* *Acinesia* (α *priv.*, and κινησις, motion,) is paralysis of motion; *anæsthesia* (α *priv.*, and αισθανομαι, I feel,) paralysis of sensibility. Paralysis has also been divided into general and partial: in the first place, when the whole body is paralysed; in the second, only a part. Partial paralysis, affecting one side, is known as *hemiplegia* (ημισος, one-half, πλησσω, I strike); when it affects the lower part of the body and extremities, *paraplegia* (παρα dim. particle, πλησσω). Local paralysis is a more limited form, an example of which may be seen in ptosis, or dropping of the upper eyelid, due to paralysis of the levator palpebræ superioris muscle from disease of the third nerve.

The following causes give rise to paralysis:—

(*a*) Disease of a nerve centre, brain, or spinal cord.
(*b*) Disease of a nerve in any part of its course.

Condition of the muscles in paralysis:—

1. Tone.—May or may not be impaired. In paralysis of *central origin* the muscles as a rule preserve their normal tonicity, but if the lesion be suddenly produced they may at first be limp and flaccid. "Early rigidity" is the result of irritation at the seat of the injury; "late rigidity" occurs in old standing cases of paralysis due to fibroid degeneration of the motor tract of the spinal cord. In *peripheral* paralysis the muscles soon become flaccid and atrophied.

2. Contractility.—Unimpaired, diminished, or lost. In "central paralysis," as a rule, electrical contractility is unaffected; but in some cases it may be exalted—in

others, impaired. The loss of contractility is in direct proportion to the amount of *muscular* atrophy. As the muscles quickly atrophy in "perepheral paralysis" their contractility is soon lost, but in a peculiar way, thus—

First Week.—Diminished alike to galvanism and Faradism *(Nerves).*

Second to Fourth Week.—Response to Faradism lost, while galvanic irritability increased *(Muscles).*

After above Period.—Muscular contractility to both is gradually but entirely lost.

The above constitutes the "reaction of degeneration."

3. NUTRITION.—Slightly, if at all, impaired.

4. ELECTRO-SENSIBILITY.—Augmented, if there is augmentation of muscular contractility; anæsthesia is not complete.

5. REFLEX ACTION.—Present in most cases.

In most cases paralysis of one side of the body, *hemiplegia*, is to be traced to disease of one hemisphere of the brain, generally to that opposite the affected side, and paralysis of the lower portion of the body and extremities; *paraplegia*, to disease or injury of the spinal cord.

HEMIPLEGIA.

Lat. *Hemiplegia.* Fr. *Hémiplégie.* Ger. *Hemiplegie.*
Syn. *Halbseitige Lähmung.*

DEFINITION.—Hemiplegia denotes paralysis of one side of the body; that is, the upper and lower extremities of one side.

In hemiplegia the muscles of the face may or may not be affected. The paralysis may be either perfect or imperfect; that is, both motion and sensibility may be lost, or only one.

This affection is generally produced by some lesion of the cortical centres, or by lesion of the motor tracts above the medulla, and may occur with or without apoplectic symptoms. The motor paths of the basal ganglia do not appear to traverse the corpora striata, but merely to pass alongside of them. The *caudate* *nucleus* is the intra-ventricular portion of the corpora striata, as the *lenticular* nucleus is its extra-ventricular portion. Between these two bodies externally lies the anterior division of the *inner capsule.* It appears that as long as a lesion of the caudate nucleus or the lenticular nucleus does not affect the internal capsule *no symptoms arise.* "Destruction of the internal capsule, however, causes paralysis of motion or sensibility, or both, on the opposite side of the body, according to the part of it which is injured" *(Stirling).* The patient suddenly falls, loss of consciousness sometimes existing for a few minutes, but from which he soon recovers. This forms the well-known "paralytic shock," or "stroke of palsy." The muscles of the paralysed limbs often become permanently contracted, or gradually waste away. The tongue is drawn towards the paralysed side, and the angle of the mouth to the sound side. The arm and leg, in severe cases, are equally paralysed; but, as a rule, the leg is less affected and recovers sooner than the arm. The lower portion of the face is also more affected than the upper.

The causes of hemiplegia are—

(*a*) The formation of a clot within the cranium, from rupture of the lenticulo-striate artery or "artery of hemorrhage" as it is called, when destruction of the lenticular nucleus may result with pressure of the internal capsule. Thrombosis, embolism, or rupture of the middle cerebral artery, causing injury to the central portion of the internal capsule, also gives rise to hemiplegia.

(*b*) The presence of an abscess in the structure of the brain.

(*c*) The growth of tumours, which may be bony, and by exerting pressure on the substance of the brain thus gives rise to paralysis.

Rigidity of the muscles betokens the continuance of irritative action within the cranium. Disease of the right side of the brain gives rise to paralysis of the left side. The face is sometimes an exception to this rule.

Disease of *right optic thalamus:* loss of sensation on left side. Injury to the posterior third in man results in disturbance of vision *(Northnagle).*

Disease of *right crus cerebri*, or *right lower half of the pons varolii:* loss of motion and sensation on left side, and facial paralysis on the same side of the lesion; if the injury is on the upper half of the pons, the facial paralysis is on the same side as the paralysis of the body *(Stirling).*

A clot pressing upon the *medulla* gives rise to derangement in the functions of respiration and deglutition.

The *faculty of speech* is impaired when the posterior part of the *left third frontal convolution* and the parts in its immediate vicinity are injured, and this fact may assist in localising the seat of the damage in hemiplegia accompanied with aphasia.

TABLE DESCRIBING THE PARALYTIC, ATAXIC, AND SPASTIC WALKS.

PARALYSIS.—In hemiplegia the patient drags the paralysed leg after the sound. The movements of the sufferer are slow, weak, halting, but uniform.

ATAXY.—The legs move as if from some unaccountable impulse, hence the patient totters and sways about, and can only maintain his equilibrium by watching the position of his legs. The body is bent forwards and the legs are apart, the advancing leg being thrown forward with suddenness and violence and brought down with a most determined stamp.

SPASTIC PARALYSIS.—The walk is a hop, the patient walking on his toes, and showing a marked tendency to fall forward.

Treatment.—Cathartics, dry cupping, unstimulating nourishment, the internal administration of the iodide of potassium, cod-liver oil, etc. When the rigidity of the muscles has subsided, but the paralysis still continues, galvanism may be tried.

TABLE GIVING THE EFFECT OF INJURY OR DISEASE TO DIFFERENT PORTIONS OF THE BRAIN.

Left Third Frontal Convolution.	Loss of power or disturbance of speech. Aphasia, may be *Ataxic*, *Amnesic*, *Paraphasia*, *Agrammatism*, *Ataxaphasia*, *Bradyphasia*.
Lenticular and Caudate Nuclei.	No permanent symptoms if internal capsule is not injured.
Optic Thalamus.	Disturbance of vision if posterior third is injured.
Internal Capsule.	Hemiplegia. Face, arm, leg, and trunk affected; recovery in inverse order *(Horsley)*.
Pedunculi Cerebri.	If one peduncle is affected, violent pain and spasm on opposite sides with contraction of the blood vessels on that side. Then anæsthesia and paresis of the *opposite* side. The oculomotorius is affected on the side of the lesion.
Pons Varolii.	Lesion of *lower half* of one side, facial paralysis of same side; and motor and sensory paralysis more or less complete on *opposite* side *(alternate paralysis)*. *Upper half* of one side, facial paralysis on same side. Sudden and extensive lesions are often accompanied with *hyper-pyrexia*. Tumours of large size, if not infiltrating, may press on the pons without giving rise to symptoms. Lesions of transverse superficial fibres often give rise to involuntary forced movements with a tendency to move to one side *(Stirling)*.
Corpora Quadrigemina.	Lesions of *anterior pair*, causes disturbance of vision, failure of the pupil to contract to light, and even blindness. Of *posterior pair*, disturbances of coördination *(Northnagle)*.

PARAPLEGIA.

Lat. *Paraplegia.* Fr. *Paraplégie.* Ger. *Paraplegie.*

Definition.—Palsy, more or less complete, of the lower half of the body, including the bladder and rectum, resulting from injury or disease of the spinal cord and its membranes. May be produced by reflex action.

Paraplegia, or paralysis of the lower extremities, is most frequently the result of disease or injury to the lower portions of the spinal cord. Very often the paralysis extends to the rectum and bladder. The power of motion may alone be lost, but not unfrequently loss of sensation is also present. In some cases sensibility is morbidly increased; this condition is known as *hyperæsthesia.* Paraplegia, as a rule, comes on gradually. It may be remarked that though voluntary motion be entirely lost, involuntary contractions of the muscles are by no means rare. These contractions may be frequently induced by irritating the soles of the feet, or tickling the inguinal region of the abdomen. Cramps frequently occur, generally at night, much to the distress of the patient. If the bladder be paralysed, the urine may be retained, and become strongly ammoniacal, or it may constantly dribble away from the patient. Another unfortunate and depressing complication is a tendency to bed-sores on the nates, due to the constant pressure on the part by long confinement in the recumbent posture.

Paraplegia has been divided into two kinds—

1. Reflex Paraplegia. This form of paralysis has been denied by some authorities.
2. Paraplegia due to Disease of the Spinal Cord.

Reflex Paraplegia may be due to—

(*a*) Intestinal worms. (*b*) Disease of the bladder and kidneys. (*c*) Pregnancy. (*d*) Teething. (*e*) Any irritation applied to the periphery of a nerve, or to any point in its course.

Proofs in favour of the theory of reflex paralysis:—

1. The cause being sought and removed, a cure is in *most* cases effected.

2. The improvement in the condition of the paralysed part, due to a like improvement or modification in the external source of irritation.

PARAPLEGIA DUE TO DISEASE OF THE SPINAL CORD.

(*a*) *Transverse section* of the *posterior columns* of the *cord* produces—

Loss of power below the section of controlling muscular movements.

(*b*) *Imperfect transverse section* produces—

1. Incomplete power of controlling muscular movements below the section.

2. Hyperæsthesia common to both cases.

(*c*) *Transverse section* of the right *antero-lateral column* causes paralysis.

On same side, and *vice versa.*

(*d*) *Complete transverse section* of *central grey matter* is followed by—

Loss of sensation in all parts below the section.

(*e*) *Transverse section* of the *left half* of the *grey matter* is followed by—

1. Loss of sensation in the right limb.

2. Hyperæsthesia of left limb, and *vice versa.*

From the above it follows that if one entire half of the cord be divided by a transverse section, there will result paralysis of motion, and hyperæsthesia on the same side as that divided, and loss of sensation only on the opposite side.

The muscles are rigidly contracted, when inflammatory action is present at the seat of disease; but they are flaccid when inflammation is absent, or has subsided.

In reflex paraplegia there is seldom an extension of the paraplegia upward, whereas in that due to disease of the cord a progressive increase is not unfrequent.

Diagnosis.—In the diagnosis of disease of the spinal cord, the reflex actions of the cord are important, as they show by their presence or absence the condition of that nervous track. For the following I am indebted to Dr Gower's book on Diseases of the Spinal Cord :—

REFLEX FUNCTIONS OF THE CORD.

SPINAL COLUMN.	REFLEX ACTION.
1 Cervical	
2	
3	
4	
5............	Scapular *(Back)*
6	
7	
8	
1 Dorsal..	
2	
3	
4............	Epigastric *(Side of Chest)*
5	
6	
7............	
8............	Abdominal *(Side of Abdomen)*
9	
10	
11	
12............	
1 Lumbar	Cremasteric *(Thigh, inner side)*
2............	
3............	Knee Reflex
4............	Gluteal *(Over Glutei)*
5............	
1 Sacral...	
2	*Ankle Clonus*
3............	Plantar *(Sole of Foot)*
4	
5............	
Coccyx	

The reflexes and the part of the cord on which they depend are thus given. The *plantar* depends upon the lower part of the lumbar enlargement; the *gluteal* on the level of the cord at the 4th and 5th lumbar nerves; the *cremaster* on the 1st and 2nd lumbar pairs; the *abdominal*, 8th to 12th dorsal nerves; the *epigastric*, on 6th to 7th dorsal nerves. These different reflexes are obtained by irritating the skin at the sole of the foot, over the glutei, on the inner side of the thigh, over the side of the abdomen, and, lastly, the side of the thorax. By their presence, increase, or absence, we have the power of determining the state of the cord in each of the divisions just mentioned, bearing in mind, however, that the "reflex excitability of the cord varies much in different individuals, is always greatest in early life, and often lessened in the old." Thus the gluteal, lumbar, and cremaster reflexes are sometimes absent in health. In cerebral disease of one hemisphere the reflexes are destroyed or lessened on the side opposite to the brain lesion. Of the deep reflexes, the "patellar tendon reflex," and the "achilles tendon reflex," are the most important. The first shows the integrity of the cord to the third lumbar nerves. It is impaired by disease—(1) of the posterior nerve roots outside the cord, or in the posterior column; (2) by disease of the grey matter; (3) of the anterior roots; (4) of the mixed trunk. It is excessive in (1) hemiplegia, on the paralysed side; (2) descending disease of the lateral columns. It is, as a rule, lost in locomotor ataxy. The achilles tendon reflex is obtained by putting the calf muscles on the stretch, by pressing the hand against the sole of the foot, the leg at the same time being extended. The ankle clonus *can never be obtained in health by sudden passive tension.*

For further most interesting information, the student is referred to Dr Gower's book above-mentioned.

Treatment.—In the treatment of reflex paraplegia, the probable cause must be sought and removed; in the other form of paralysis, counter-irritation to the spine, and the free administration of tonic medicines will be required. In the presence of inflammatory action around or in the cord, strychina should not be given, but preparations of Calabar bean may be used together with the iodide of potassium.

Table showing Points of Distinction in Hemiplegia, Paraplegia, and Locomotor Ataxy, as regard Cause and Symptoms.

	HEMIPLEGIA.	PARAPLEGIA.	LOCOMOTOR ATAXY.
Causes.	Rupture of a blood-vessel in the brain. Destruction of the basal ganglia. Disease of brain opposite to side affected.	Disease or injury to the spinal cord.	Wasting disease of posterior columns of the spinal cord; formation of amyloid corpuscles, and increase of connective tissue. Disease of the cerebellum.
Symptoms.	Loss of voluntary power over the muscles. Motion reflexly induced may, however, still remain. Emotional movements also do not suffer. Movements of the eye, neck, back, and chest are not impaired. Movements of the chest alike on both sides. Affected lip drawn to healthy side. Cheek hangs flabby, like a sail with but light wind to blow it out. Tongue generally drawn to affected side from paralysis of the genio-glosus muscle. Paralysis of the *arm* most marked.	Loss of muscular power in lower limbs more or less complete. Loss of power depending on part of cord injured. "If the disease be seated high up, as in the cervical or upper part of the dorsal region, there is usually difficulty in the act of micturition, owing to spasms of the sphincters; if, on the other hand, the disease be situated in the lower dorsal or lumbar region, the sphincters are paralysed, and the urine consequently escapes spontaneously." Thus loss of motion without loss of sensation, loss of sensation without loss of motion.	Muscular power diminished, but still present; total loss of the power of co-ördinating movements. Difficulty in walking or in regulating the movements of the lower limbs. May be a symptom of poisoning by alcohol, lead, and mercury, or of disease of the cerebellum. The patient may be able to walk as long as he keeps his eyes on his feet, but falls immediately he looks up.

Table showing the Symptoms which help to distinguish Facial or Bell's Palsy of the Seventh Nerve from Palsy due to Cerebral Disease.

	FACIAL PALSY, DUE TO SEVENTH PAIR OR FACIAL NERVE.	PALSY OF THE FACE, DUE TO CEREBRAL DISEASE.
Cause.	1. Exposure to cold, disease of the parotid gland, injury to the nerve in any part of its course from pressure in the bony canals, through which it passes. Inflammation of the nerve in the aqueductus Fallopii, generally the result of cold. Disease in the floor of 4th ventricle. Disease of any of the bones in the ear.	1. Disease of the brain, opposite to the side affected.
Symptoms.	2. Paralysis of muscles supplied by the facial nerve complete. Want of power to close the eye, even when asleep, due to paralysis of the obicularis palpebrarum. Loss of power to kiss or whistle, due to paralysis of orbicularis oris and buccinator. Patient cannot frown; all wrinkles disappear. The masseter, temporal and pterygoides, act well. Electrical contractility of muscles lost.	2. Paralysis of face incomplete. Movement of the eyelids unaffected. Patient can close his eye and is able to whistle. Patient can frown, though not so well as in health. Wrinkles of the forehead do not entirely disappear. Paralysis of the temporal masseter, and pterygoides due to implication of the fifth pair. Electrical-contractility of muscles present.
Prognosis.	3. Will depend upon the cause. If from cold, it may soon pass off. If the electro-contractility in the muscles but slightly impaired, the prognosis favourable.	3. Unfavourable.
Treatment.	4. Hot fomentations, leeches, blisters, tonics. Faradisation.	4. The treatment before given for paralysis.

TABLE GIVING THE RESULTS OF PARALYSIS AFFECTING SPECIAL CRANIAL NERVES.

First Nerve. Olfactory.	Anosmia, with some loss in the sense of taste. Apart from nerve disease, loss of smell may be due to diseased conditions of mucous membrane of nose, polipi, catarrh, etc.
Second Nerve. Optic.	Dimness of vision, due to atrophy, or optic neuritis. May be a symptom in Bright's disease, ataxy, cerebral tumours, hydrocephalus in children.
Third Nerve. Motores Oculorum.	Divergent squint, as all the muscles of the eye except the superior oblique and rectus externus are supplied by it. Ptosis due to paralysis of levator palpebrarum, dilatation of pupil.
Fourth Nerve, or Pathetici.	Absence of rotatory power of eye, diplopia, pupil slightly above lower lid. If left side affected, patient, when looking at objects in front of him, turns his head downwards and to the right.
Fifth Nerve. Trigeminal.	*Ophthalmic Division.*—Loss of sensation over part of the forehead, upper eyelid, or mucous membrane of nose. *Superior Maxillary Division.*—Loss of sensation on one side of the face, lower eyelid, cheek, side of nose, upper lip, gums, etc. *Inferior Maxillary Division.*—Lateral part of head, with anasthesia of those parts, lower lip, half of tongue, muscles of mastication paralysed.
Sixth Nerve. Abucentes.	Paralysis of external rectus with convergent squint.
Seventh Nerve. Motor Branch of Auditory.	Bell's or facial palsy. (See Table, p. 426.)

PARALYSIS AGITANS.—SHAKING PALSY.

Lat. *Paralysis agitans.* Fr. *Paralysie tremblante.*
Ger. *Paralysis agitans.*

Definition.—A disease of the nervous system, characterised by trembling of the muscles. The muscles are in a perpetual alternation of contraction and relaxation independent of the will.

Paralysis agitans may be the result of exposure to cold, or may follow violent fits of mental emotion, fear, or rage.

It is generally a disease of advancing years, seldom occurring before forty.

Symptoms.—The disease may come on suddenly in the muscles of one hand, or its appearance may be gradual, attracting little attention for some time. The movements are convulsive, and are rendered worse if the unfortunate sufferer feels that notice is being taken of him. In standing, a stooping attitude is assumed, the trembling movements continuing in spite of all efforts made by the patient to arrest them. His gait is peculiar, and almost laughable were we not reminded of the unfortunate condition of the patient. Thus, he will rise from his chair, try to steady himself, and, on attempting to walk, start off into a run, in many cases ending in a fall. I remember once seeing a patient who, when requested to stand with his back close against a wall, had to be removed owing to the blows he gave his head during the convulsive movements of his whole body, which were rendered worse when he perceived that any one was noticing him. During the course of the disease the muscles may become rigid and contracted, these conditions being accompanied with cramps and neuralgic pains. Of the muscles which become contracted, the flexors suffer most.

Morbid Anatomy.—The pathology of paralysis agitans is obscure, and the changes found after death in the nervous system are not characteristic or permanent. Charcot has found obliteration of the central canal of the cord by overgrowth of the epithelial lining, and pigmentary deposit in the nerve cells of the cord, especially in the posterior vesicular columns of Clarke.

Prognosis.—Unfavourable.

Treatment.—Very little can be done by way of treatment. Strychnia has been recommended by Trousseau, but condemned by others. Improve the general health.

TABLE CONTRASTING THE FOLLOWING DISEASES.

	INFANTILE PALSY.	DIPHTHERITIC PALSY.	SATURNINE PALSY.	PROGRESSIVE MUSCULAR ATROPHY (WASTING PALSY).	LOCOMOTOR ATAXY.
Cause.	Obscure. Teething, eruptive fevers. Exposure to cold.	The result of a specific poison, causing the primary diphtheria.	Chronic lead poisoning.	Obscure. Exposure to cold. Mental and bodily exertion. Hereditary taint.	Obscure. Exposure to wet (?) Sexual excesses (?) Hereditary tendency, etc. etc.
Symptoms.	Invasion sudden, attended in some cases with febrile movement, convulsions, coma, etc. The muscles rapidly become paralysed; in many cases only two days may elapse from the onset of the disease. The legs and arms may be attacked, or the legs only, or the arms only. The muscles	Comes on as a sequel to diphtheria. In many cases the paralysis affects only the soft palate, and is apparently not due to local inflammatory changes. Thence it may proceed to other muscles, the patient complaining of numbness and tingling, and loss	In most cases the paralysis begins in the extensors of the right forearm, especially those supplied by the radial nerve. An attack of colic in most cases precedes the paralysis. The forearm or the fingers cannot be extended. The flexor muscles	Invasion insidious. Absence of any marked constitutional disturbance. The attention may be first drawn to a loss of power of some muscles or group of muscles. The atrophy of the muscles may commence at any part, but most frequently in the right hand, "whence it spreads to the corresponding muscles of the opposite side of the body, then to those	Muscular power diminished, but still present; total loss of the power of co-ördinating muscular movements. Difficulty in walking, or in regulating the movements of the lower limbs. The patient may be able to walk, as long as he keeps his eyes on his feet, but falls immediately he looks up. The patient may suffer from pain of a

Symptoms—Continued.	become flaccid, and reflex excitability is impaired or entirely lost. An *important* symptom is the rapid impairment, or absolute abolition, of electrical contractility, so that in four or five days the muscles may have entirely ceased to contract under the influence of Faradisation. Hyperæsthesia may also be present, but, as a rule, there is total absence of pain, "Indeed, it may be regarded as characteristic of infantile paralysis that absolute paralysis of certain muscles, attended with flac-	of power in one or both lower extremities, and then the upper extremities may be alike affected. A general paresis may next supervene. In other cases the paralysis may spread from the fauces along the muscles of mastication, of articulation, and of expression. There may be squinting and double vision, and in some cases temporary amaurosis. Sexual power and appetite may be entirely lost. The paralysis is in most cases pro-	still retain their power, and the fingers are therefore bent on the palm. The muscles waste rapidly, but, whilst losing their electro-contractility, retain their electro - sensibility. Cutaneous sensibility is not impaired. The following is the order in which the muscles are, as a rule, attacked:—The extensor communis digitorum, the extensor indicis, the extensor minimi digiti, the extensor secundi internodii pollicis,	of both forearms, and presently becomes distributed with more or less irregularity, but symmetrically, throughout the trunk and lower extremities." The muscles of the ball of the right thumb are those commonly first affected. The progress of the disease may vary from the course given above. It may be distinguished from acute spinal paralysis. *First.* The atrophy precedes the enfeeblement of the affected muscles. Entire loss of motion only occurs when the muscles are completely atrophied. *Second.* The electro-contractility of the affected muscles remains unimpaired, and only disappears when	very varying character, sometimes acute, sometimes dull, at other times of a stabbing, darting character. The patient may suffer from squint, and a difficulty in distinguishing colours, or from deafness in one or both ears. An important feature in this disease is the affections of the joints, which consist in a rapid effusion into them which may gradually pass off, or end in total destruction of the joint. According to Duchenne, "the muscles of the affected limbs retain, as a rule, their bulk, their tonicity, their electrical contractility, and their

[TABLE—*Continued.*

TABLE CONTRASTING THE FOLLOWING DISEASES—*Continued.*

	INFANTILE PALSY.	DIPHTHERITIC PALSY.	SATURNINE PALSY.	PROGRESSIVE MUSCULAR ATROPHY (WASTING PALSY).	LOCOMOTOR ATAXY.
Symptoms—Continued.	cidity and loss of reflex and electrical contractility is linked with an almost total absence of pain, with retention of cutaneous sensibility, perfect control over the rectum and the bladder, and a total absence of all tendency towards inflammation of the urinary organs, the formation of bed-sores, or the appearance of other cutaneous inflammations."	gressive, and has a tendency to shift from one part to another. Sensation & motion are usually simultaneously affected, but the loss of sensation is greater than the loss of motion, and the paralysed muscles retain their electric contractility, their size, and their healthy structure.	the extensor carpi radialis brevior, the extensor carpi radialis longior, the extensor carpi ulnaris, the extensor ossis metacarpi pollicis, and the extensor prima internodii pollicis. (*Duchenne.*) Sometimes the deltoid is alone affected, or the muscles of the lower extremities, and then chiefly the extensors.	the muscular fibres are entirely atrophied. *Third.* "A very common feature of the malady is the presence in the affected muscles of irregular vibratile movements of the individual fibres, which may be both seen and felt." These movements are not peculiar to this disease. Control over the sphincters is not lost as a rule. There is absence of spinal pain, and bed sores are not readily formed. Owing to an excess of cutaneous fat, the atrophy of the muscles may for a time be disguised.	strength, little, if at all impaired, not only so long as the patient can walk or stand, but long after his limbs have become absolutely helpless. And often at a time when the patient cannot rise from his chair or stand, he can freely execute movements of extension and flexion as he sits or lies, and successfully resist all manual efforts on the part of others to extend or flex his legs."

Prognosis.	Favourable.	Favourable.	Favourable, if treatment be adopted early.	Unfavourable.	Unfavourable.
Morbid Anatomy.	Atrophy and sinking of the muscles, disease of the spinal nerves and anterior cornua of the cord, with a deposit of pigment in the part, and atrophy of the nerve cells, with more or less sclerotic change.	Obscure. Medulla oblongata is in some cases affected, but the nature of lesion is not clear.	Muscles pale and yellowish, shrunken, but otherwise healthy, unless the disease has lasted for some time, when degenerative changes may take place. The palsy is probably due to disease of the nerve trunks, or of the nerve cells, from which the nerves take their origin.	Changes more or less present in the motor nerves, and in the anterior cornua of the grey matter of the cord, probably of inflammatory origin. The muscular structures become attenuated, and, as the disease progresses, fatty.	Sclerosis of the posterior columns of the spinal cord. But, according to Charcot, a small amount of disease in the cord will produce all the symptoms of locomotor ataxy.

[TABLE—*Continued*

TABLE CONTRASTING THE FOLLOWING DISEASES—*Continued.*

	INFANTILE PALSY.	DIPHTHERITIC PALSY.	SATURNINE PALSY.	PROGRESSIVE MUSCULAR ATROPHY (WASTING PALSY).	LOCOMOTOR ATAXY.
Treatment.	In early stages, expectant. Regulation of the bowels, remove worms, etc. Faradisation.	Faradisation. Nux vomica, strychnia, friction.	The treatment of lead colic as before mentioned. Faradisation for the palsy, each muscle being separately galvanised for ten or fifteen minutes at a time.	Fadarisation applied to each muscle in turn. Improve general health; tonics, change of air, etc.	Rest, avoidance of excessive exertion. Tonics, iron, nitrate of silver, iodide of potassium, etc. Faradisation. Wet sponging

DISEASES OF THE SPINAL CORD.

ACUTE SPINAL MENINGITIS.

Lat. *Meningitis spinalis.* Fr. *Méningite spinale.*
Ger. *Entzündung der Häute.*

Definition.—Inflammation of the membranes of the Spinal Cord.

The membranes of the cord may become the seat of acute inflammation.

Symptoms.—The symptom which marks the presence of the disease is, pain of a burning character felt down the spinal column, and darting into the extremities. The slightest movement of the body increases the pain, and also pressure along the vertebral column. Opisthotonos, due to the powerful contraction of the muscles of the back, may also occur. Paralysis, caused by pressure on the cord from the effusion of fluid under the membranes, is not unfrequent. The disease, in most cases, ends fatally.

Treatment.—The treatment will consist in the adoption of the general measures recommended when speaking of acute cerebral meningitis. When the disease becomes chronic, the application of the actual cautery may be of use.

MYELITIS.

Lat. *Myelitis.* Fr. *Myélite.* Ger. *Entzündung des Marks.*

Definition.—Inflammation of the substance of the Spinal Cord.

Inflammation of the structure of the spinal cord is very rare, and has no well-marked symptoms which may be considered characteristic. A certain amount of paralysis always accompanies the disease. The part of the body paralysed will depend upon the portion of the cord in which the inflammatory process exists. The cord may become softened, and suppuration, the result of the inflammation, ensue. Hæmorrhagic extravasation is not unfrequent, and is considered by Charcot to be in most cases the result of inflammatory softening. The term *sclerosis* is often applied to grey softening of the cord. This disease, the result of inflammation, is considered by Charcot to begin in the nerve cells. Sclerosis has a marked tendency to be limited to certain portions of the nervous centres, thus giving rise to a variety of symptoms which depend upon the part attacked. Hydatid cysts may form in the cord; it may also be the seat of tubercle, congestion, etc.

Hæmorrhage between the membranes of the cord, giving rise to what is called *spinal apoplexy*, is very rare. When it does, however, occur, it constitutes one of the morbid conditions which enter into the causation of paraplegia and general paralysis.

Spinal irritation, marked by tender spots over several parts of the spine and neuralgic pains in the back, is said to be due to anæmia of the posterior columns. It frequently occurs in delicate women. The hypodermic injection of strychnia is said to cure this disease, thus distinguishing it from other diseases of the cord.

ACUTE ASCENDING PARALYSIS.—"LANDRY'S" PARALYSIS.

Lat. *Paralysis ascendens acuta.* Fr. *Paralysie aiguë ascendante.* Ger. *Acute aufsteigende spinalparalyse.*

DEFINITION. —A disease characterised by motor paralysis attacking the lower extremities first, and more or less rapidly extending to the upper extremities and trunk and to those parts connected with the cervical enlargement of the cord and medulla oblongata. The disease is more frequent in men than in women, and generally occurs between twenty and forty years of age.

Both the etiology and morbid anatomy of this curious, and happily rare, affection are unknown. It has been attributed to exposure to cold and wet, and of course to syphilis, that unhappy disease from which each obscure affection is now held to take its origin. Morbid anatomy is equally silent as to the nature of the affection, for with the exception of one or two cases, the correct diagnosis of which have been doubted, the spinal cord, medulla oblongata, brain, nerves, and muscles have showed no signs of disease. Inflammatory changes in the central cells of the anterior cornua of the cord have been noticed, but these changes are not constant. By Landry, it was considered to be due to some toxæmic material in the blood, closely allied to that probably existing in the infectious fevers; the enlarged spleen, and other glandular organs, lending some support to this view. Others have suggested that the disease may be a modified form of general bulbar and spinal inflammation, in which the medulla and lateral columns are specially implicated.

Symptoms.—The early symptoms are somewhat obscure. Slight febrile disturbance, accompanied with pains in the lower extremities lasting but a few days, as a rule, mark the onset of the disease. Muscular weakness, quickly ending in complete paralysis, is the most marked symptom. The paralysis begins in the muscles of the toes and feet, proceeding to those of the leg and thigh, and then travels up to the hands, arms, and trunk; ending in the death of the patient from starvation, or asphyxia from implication of the muscles of deglutition and respiration. The muscles do not exhibit any marked atrophy, and do not present the "reaction of degeneration." The reflexes are for a time retained, but soon become diminished or lost. Sensory troubles are slight, and the bladder and rectum are seldom affected; there are no cerebral symptoms, and trophic changes in the skin do not occur. During the course of the attack, which may last from eight days to three weeks, the average being twelve days, the general state of nutrition is not interfered with, although there may be occasional attacks of febrile disturbance.

Diagnosis.—The following Table will aid in the recognition of this very fatal disease:—

LANDRY'S PALSY.	ACUTE ASCENDING MYELITIS.	POLIO-MYELITIS ANTERIOR ACUTA.
Absence of sensory derangements and trophic changes in the skin. Bladder and rectum unaffected. Muscle after muscle is attacked. Absence of "reaction of degeneration."	Disturbance of sensibility, the bladder and rectum suffer, and bed sores appear.	Muscles rapidly waste. Faradic excitability early lost. "Reaction of degeneration" well marked. Absence of bed sores and bladder and rectal troubles.

Treatment.—At first expectant, with attention to the bowels, etc. Belladonna, ergot, iodide of potassium may be tried.

DISEASES AFFECTING THE URINARY ORGANS.

NEPHRITIS.

Nephritis (Νεφρος, *the kidney terminal itis,*) is an acute inflammation of the substance or parenchyma of the kidney, rarely met with.

PYELITIS, inflammation of the mucous membrane of the pelvis of the kidney, though also a rare disease, is more frequently met with than nephritis, to which it generally gives rise by an extension of the inflammatory action.

Pyelitis may result from the irritation caused by the presence of a stone in the pelvis of the kidney, or by the impaction of a calculus in the ureter, giving rise to distension of the pelvis and calices of the kidney.

The usual causes instrumental in the production of nephritis are, exposure to sudden cold, the abuse of alcohol, and the careless administration of oil of turpentine and the tincture of cantharides. Sometimes a fly blister applied in the region of the kidneys will give rise to the disease.

Inflammation of the kidney is occasionally met with in strumous, ill-fed persons, in whom it may arise without any appreciable cause.

Symptoms.—The following may be taken as the history of a typical case of this disease:—A man addicted to habits of intemperance leaves a hot ill-ventilated room, and elects to pass the greater part of the night in the street. A day or two after he complains of having had a shivering fit, and is troubled with nausea and vomiting. His skin is hot, his pulse, is hard, full, and frequent, and his thirst is incessant; pain is felt over the region of the kidney; there is tenderness on pressure and retraction of the testicle on the side affected; the urine is high-coloured, scanty, or suppressed if both kidneys be affected. The desire to void urine is frequent and urgent. Diarrhœa and vomiting are not unfrequent. When pyelitis is also present, the urine contains muco-pus in more or less abundance. The inflammation may end in resolution, or it may go on to suppuration and the formation of abscesses. Even under the most favourable circumstances, the kidney is generally left enlarged, and more or less indurated. The future health of the patient will depend upon the condition in which the kidneys are left after the acute attack has passed off.

Diagnosis and Prognosis.—Acute nephritis must be distinguished from lumbago, perinephritis, spinal disease, and the passage of a renal calculus from the kidney to the bladder. The first may be known by the absence of any marked constitutional disturbance. The urine is either clear or loaded with urates, but there is no albumen or renal tube casts. In perinephritis and spinal disease, the symptoms come on more gradually; the bladder is not irritable, the testicle is not retracted; and, in the last disease, careful examination along the spinal column will detect pain at one particular spot on the application of pressure. In the case of the passage of a renal calculus, pain comes on suddenly, shoots downwards from the kidney to the bladder, and may as suddenly cease as it came on. Constitutional symptoms are present, are more marked, and approach nearer those of nephritis.

The *Diagnosis*, therefore, requires care, as the treatment varies in each case.

The *Prognosis* will depend upon the severity of the inflammatory process, and the nature of the probable cause.

Uræmia, should it occur, will greatly add to the danger, and lessen the chance of recovery.

Treatment.—Local depletion, if necessary. Dry cupping has in some cases afforded relief. Hot fomentations and poultices should be applied over the loins. Hot hip baths, or hot air baths, may be employed with advantage. To relieve the pain, Dover's powder may be given at night. The diet at first should be low, but should symptoms of prostration make their appearance, a more nourishing diet must be allowed. Purgatives and diaphoretics are indicated, in order to relieve the kidneys as much as possible of their work. Rest in bed and perfect quiet will make up the necessary treatment.

BRIGHT'S DISEASE.—SYN. ALBUMINURIA.

Lat. *Morbus Brightii.* Fr. *Maladie de Bright.*
Ger. *Bright'sche Krankheit.*

DEFINITION.—A generic term, including several forms of acute and chronic disease of the kidney, usually associated with albumen in the urine, and frequently with dropsy, and with various secondary diseases, resulting from deterioration of the blood.

Albumen may be present in the urine during the course of certain fevers and other acute diseases; also in the advanced stages of valvular disease of the heart, and in cases of severe emphysema, complicated with bronchitis; but then its presence is, as a rule, only transitory, and is not due to any permanent structural derangements of the kidney. The albuminuria present in the course of valvular disease of the heart, and in any form of lung disease which prevents the proper circulation of the blood, is the result of the passive venous congestion of the kidney, due to the impeded venous circulation. Above all, it must be remembered that in some very rare cases of Bright's disease albumen is sometimes absent from the urine *(Johnson).*

Pathology.—The origin of Bright's disease must be sought for in the blood. Although we may never be able to fully appreciate the exact condition of the blood which gives rise to the pouring out of albumen in the urine, and to the ultimate structural changes which take place in the kidney, yet from the following facts we may safely refer the diseases about to be described to a morbid condition of that fluid. Bright's disease attacks both kidneys; the disease is therefore bilateral, the exceptions to this rule being few and far between. The character of the morbid appearances is in most cases exactly alike in both kidneys, although in one kidney the disease may be more advanced than in the other. As further proof of the blood origin of this disease, it may be observed that, in most cases, it occurs concurrently with, or as a sequel to, certain diseases in which a diseased condition of the blood may be confidently predicated. The intimate connection between scarlet fever and acute Bright's disease will be dwelt upon more fully when speaking of the latter disease. Dr Johnson is of opinion that the blood, in its morbid condition, "exerts its influence primarily and chiefly upon the gland-cells which line the convoluted tubes. That the cortex or secreting portion of the kidney is the seat of the disease, while the medullary cones, even in the advanced stages of the malady, are left comparatively intact." By the aid of the kidneys the blood is purified of certain morbid and excrementitious matters, and it is probably during the exercise of the healthy function of the glands that a heaping-up and subsequent arrest in the secreting cells of the morbid materials circulating in the blood take place, which lead to the structural changes in the kidneys. These structural changes take place in the malpighian capsules, and in the cells of the basement membrane of the tubes. Virchow has, according to Dr Johnson, mistaken these changes for a formation of connective tissue *between* the tubes. In the course of time, and as the disease progresses, the blood-vessels themselves undergo certain changes—blood escapes, and comes away in the urine, together with the inner lining of the uriniferous tubes, known as *tube casts.* Fat globules, the result of a fatty degeneration of the gland, also make their appearance in the urine, in the more advanced stages of the disease.

ACUTE BRIGHT'S DISEASE.

Lat. *Morbus Brightii acutus.* Fr. *Maladie de Bright aiguë.* Ger. *Acute Bright'sche Krankheit.*

Syn. *Acute albuminuria, Acute desquamative nephritis, Acute parenchymatous nephritis, Acute renal dropsy.*

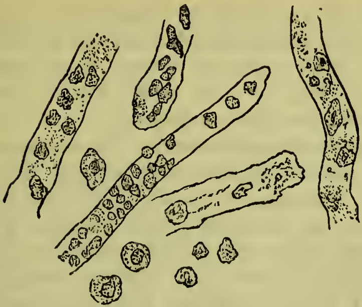
As a sequel to scarlet fever, acute desquamative nephritis is not unfrequent. It then generally follows mild attacks of that disease, and is due to the exposure of the patient to cold during the stage of desquamation. The burden of the removal of the poison is therefore thrown on the kidneys, which are unable to bear the strain. It has been frequently remarked that, during an epidemic of scarlatina, there is a strong tendency for simple diseases—catarrh, simple sore throat, etc.—to assume a diphtheritic or croupous type. It is not improbable, therefore, that the kidneys become more or less affected, and that when to this condition the poison of scarlatina is added, the result is the disease in question.

When acute desquamative nephritis does not follow an attack of scarlet fever, it may arise from other causes, as exposure to cold, intemperance, and as a sequel to diphtheria, measles, erysipelas, and cholera, especially during the typhoid stage of the latter. Excessive indulgence in the use of alcohol is more likely to give rise to chronic than to acute Bright's disease. Dr Johnson, however, mentions a case of a man brought to the hospital one night, and believed to be drunk. His stomach was emptied of a large quantity of spirituous fluid, but as he did not recover, and uræmic symptoms made their appearance, some of his water was drawn off, and found to be "loaded with albumen." He was cupped over the loins, and a purgative administered. Next morning he was quite sober, and his water perfectly free from albumen. In this case the transient albuminuria was probably due to congestion of the kidneys, due to the extra strain placed upon them while excreting the excess of alcohol.

Symptoms.—Perhaps suddenly during the latter stages of the desquamative process in scarlet fever, that is, between the seventeenth and twenty-third day after the subsidence of the eruption, a rigor, followed by vomiting, may mark the accession of the renal complication. In some cases, however, you will find that a puffiness under the eyes, and a marked pallor of the face, not unlike the peculiar colour of boiled pork, is first noticed by the friends of the patient. In others, especially in young children recovering from scarlet fever, and whom you may not deem it necessary to see every day, you may be suddenly called to see the child in "a fit." The child's countenance will immediately give you a clue to the true state of things, rendered beyond doubt on examining the urine and finding it albuminous. The presence of vomiting is an important *diagnostic* symptom. The pulse is quick and throbbing, the thirst great, the appetite entirely lost, and pain is felt in the loins, and sometimes down the inner side of the thighs and in the testicles. The skin is hot and dry. The patient also makes frequent attempts to pass water, especially at night, when in the recumbent posture.

The quantity secreted is small, ten to sixteen ounces only being passed in the twenty-four hours, or the secretion may be entirely suppressed; of high specific gravity (1030–1065), albuminous, of a smoky or of a dirty brownish colour; this latter condition being most characteristic of acute Bright's disease, and is due to the admixture of blood. The urine is generally acid. Inflammation of the peritoneum and of other serous membranes may occur in some cases, and dropsical effusion into the pleura and pericardium is not uncommon. The lower lobes of the lungs may become œdematous, and thus greatly impede respiration. The patient may have several convulsive attacks, from which he may recover, or coma may supervene, and he may die comatose. A gradual return of the urinary secretion to its normal healthy amount marks the advance to convalescence.

The Urine.—If the sediment be examined under the microscope, it is found to be composed of casts from the minute urinary tubes—*epithelial casts*—and of other casts, probably of coagulated fibrin—*hyaline* or *waxy casts*—generally having a granular appearance, from the presence of a multitude of epithelial cells.



Epithelial casts commonly present in the urine in cases of acute dropsy *(Beal).*

Blood corpuscles, in varying stages of disintegration are also present. Some of the casts consist of coagulated blood and fibrin, and are known as "blood-casts." The hyaline casts vary in size, the smaller being formed within the uriniferous tubes not yet denuded of the gland-cells; the larger are from those tubes where the exfoliation of the gland-cells has taken place. In the acute form of the disease the smaller hyaline casts are by far the most numerous. Sometimes also oily cysts and cells make their appearance in the urine, and point to the presence of a state of fatty degeneration going on in the gland-cells. The urine should always be examined both before and after a meal, and after exercise, to get a fair average of the amount of albumen present.

The Blood.—As a result of the unnatural drain through the kidneys, the blood rapidly deteriorates, and its water is increased, whilst there is marked diminution of the albumen, with increase of urea and uric acid. The surface of the body assumes an anæmic appearance, due partly to the diminished number of the blood-cells as the disease proceeds, and partly to the amount of water in the subcutaneous tissue. When drawn from the body the blood displays a buffy coat, due to excess of fibrin. The inorganic salts and fat are not affected. The density of the serum is reduced from 1030 to 1019, and this reduction is most marked when the quantity of albumen in the urine is largest.

Post-mortem Appearances.—The appearance which the kidney presents is that of hyperæmia and incipient exudation. The kidney is enlarged, soft, easily torn, and of a more or less dark brown colour. The capsule is readily removed. On section, numerous ecchymoses are seen scattered about the surface, which is more or less covered by an adhesive fluid, the result of the inflammatory process. The cortical is increased at the expense of the pyramidal portion. The uriniferous tubules are found full of epithelium, and the malpighian capsules engorged with blood. The accumulation of epithelial cells in the uriniferous tubes may give a whitish or mottled appearance to the organ.

Under the microscope the convoluted tubes are found more or less choked up with epithelium in varying stages of disintegration and degeneration, oil and blood-cells being found here and there in the general destruction. The minute capillaries are in some cases found ruptured, with escape of the blood into the neighbouring parts.

The primary cause of this rapid disorganisation of the kidney is, probably, the result of an abnormal demand on the gland in the due performance of its physiological function of excreting from the blood certain deleterious products. If the material in the blood, of the nature of which we are entirely ignorant, be in small quantities, no mischief to the kidneys may result; but when there is an excess and a constant and prolonged demand made upon the physiological functions of the gland, disease is set up; the morbid phenomena are, therefore, the result of a modified physiological function. Should the morbid materials be rapidly removed or become decomposed in the blood into harmless compounds, the gland gradually recovers its former healthy condition, and its physiological functions are regained. The general febrile state of the patient is the result of the presence of urinary constituents in the blood.

Diagnosis and Prognosis.—The diagnosis is only difficult in those cases of acute desquamative nephritis unattended by dropsy; but the diagnosis becomes more difficult when we are required to decide whether the case before us is a recent or chronic attack. The history of the case is important, and the colour of the urine and the character of the sediment will all assist us in the formation of our opinion. In diphtheria, the albuminuria is seldom attended with dropsy.

Œdema, diagnostic of renal disease, appears first in the face, especially about the eyelids, in the morning, the ankles and scrotum becoming swollen towards evening. The face is puffy, and assumes a pasty look. Anasarca of the hands and legs next occurs. The dropsical effusion has a marked tendency to shift from one part of the body to another.

In the acute forms of the disease, the prognosis is favourable, especially if the patient come early under treatment. In the young and the middle-aged the disease is more amenable to treatment than in the aged; recoveries are therefore more frequent among the former than among the latter. Any symptoms which betoken head complications must be regarded with suspicion. Inflammation of the endocardium and pericardium are also important and dangerous concomitants. A return to the normal amount and constitution of the urine marks the improvement in the condition of the patient; but recovery may be prolonged for months, and, as long as the urine remains albuminous, a cure cannot be said to have been effected. If the albuminuria be the result of a specific poison—scarlet fever—or of exposure to cold, there is more probability of ultimate recovery than when there is a constitutional taint, of the nature of which we are ignorant.

TABLE AS AN AID TO DIAGNOSIS BETWEEN ACUTE AND CHRONIC BRIGHT'S DISEASE.

ACUTE.	CHRONIC.
Urine high-coloured, smoky, and blood-tinged. Sediment copious, consisting of epithelial and blood-casts. Sometimes a few oily cells and casts. Sp. gr. high.	Urine pale-coloured, highly albuminous; oily casts and cells; epithelial casts absent, or nearly so. Large hyaline casts. Sp. gr. low.

Treatment.—Dr George Johnson recommends the rigid employment of a milk diet, and he has shown that a change of the diet from milk to fish will increase the amount of albumen in the urine. He also recommends that the urine be tested *after* meals for several days, and if under these circumstances the urine remains free from albumen, the cure may be considered perfect.

Indications for treatment:—1. *To arrest the inflammatory action.* 2. *To give rest to the kidney.* 3. *To remove the dropsy.*

1. In robust and plethoric individuals, local bleeding by cupping or leeching over the region of the kidneys may be employed. Hot poultices applied across the loins are also found useful.

2. Diaphoretics and hydragogue purgatives, to produce a free action of the skin and bowels, and thus relieve the kidneys of a portion of their work, will be necessary. The hot air bath will be found a most valuable adjunct to other means adopted. General bleeding and the use of calomel are contra-indicated. Patients suffering from acute Bright's disease show a marked susceptibility to the action of mercury, profuse ptyalism not unfrequently following the administration of even small doses of the drug.

3. To remove the dropsy, the compound jalap or the compound scammony powder will be the best for the purpose; in severe cases, elaterium must be used. The

tincture of steel given during this stage is often attended with marked improvement in the condition of the patient. Sir Robert Christison recommends the use of digitalis in combination with the acid tartrate of potash. Dr Roberts suggests the use of the citrate of potash and broom tops, in any stage of the disease, to reduce the dropsy.

During convalescence the patient should be warmly clad, and exposure to cold scrupulously avoided. Flannel should be worn next the skin.

CHRONIC BRIGHT'S DISEASE.

Lat. *Morbus Brightii longus.* Fr. *Maladie de Bright chronique.* Ger. *Chronische Bright'sche Krankheit.*

Syn. *Chronic Albuminuria.*

Chronic Bright's disease of the kidney is now considered to depend upon a primarily morbid condition of the blood. "May it not be possible that by some failure in preparation, either by the stomach or liver, albumen enters the blood on the right side of the heart in a still crude state, and in a condition similar to that in which it would have been had it been introduced through the jugular vein; and thus, being unassimilatable, is it not excreted, as in Bernard's experiment, by that ready outlet, the kidney? In support of such a supposition, we have the facts, that many cases of kidney disease seem to be most probably of blood origin, and that among the common antecedents of Bright's disease are circumstances of diet or mode of living which would be likely to impair the processes which should go on in the stomach or liver" *(Parkes on the Urine).*

In the course of the remarks about to be made on the subject of chronic Bright's disease, certain lesions of the kidneys will require detailed notice. By some these lesions are considered to represent different stages of the same disease. There are others, however, who entertain contrary opinions. The following is a summary of some of the views expressed on this subject:—"An engorgement of the renal blood-vessels, an effusion of inflammatory products, a more or less complete and general metamorphosis of these products into fat, and, finally, atrophy and wasting of the kidneys. The small contracted kidneys have once been fatty; the large, pale kidneys are in constant progression towards atrophy and contraction."

Dr Johnson does not entertain this view, for he maintains that in the case of the small red granular kidney there has never been any enlargement of the gland, "but from the commencement a process of wasting occurs." Some authorities also maintain that the large white kidney is the second stage of *acute tubal nephritis*, and that there is a third stage, contracted granular kidney of tubal origin, different from the contracted granular or gouty kidney due to chronic interstitial nephritis. The clinical histories of contracted kidney, and of the large white kidney, confirm the view of their being distinct diseases.

These lesions are as follow:—

1. GRANULAR KIDNEY. Synonyms—*Contracted granular*× *Kidney, ~~Chronic desquamative~~*+ *nephritis, Gouty Kidney.*—The kidneys in this case are greatly reduced in size, and may each only weigh an ounce and a half. The cortical portion, which appears to be that portion most affected, is notably diminished, the medullary cones extending almost to the surface of the kidney. The structure of the organs becomes dense and hard, and the capsule so adherent that it cannot be removed without portions of the gland attached to it. The organ, even in the most advanced stage of the disease, retains its normal red colour. Dr Grainger Stewart considers that the diseased process commences in the intertubular matrix, and that it consists in an hypertrophy of the fibrous stroma of the organ, which destroying the uriniferous tubes and malpighian bodies,

× or Cirrhotic
+ Interstitial

causes a contraction and reduction in the size of the organ. Dr Johnson, while controverting the doctrine of Virchow, that there are three anatomical elements in the kidney, namely, tubes, vessels, and interstitial tissue, and that it is to the diseased condition of one or more of these tissues that the varieties in the appearance of the kidney in chronic Bright's disease are due, remarks—"My doctrine with regard to the minute anatomy and pathology of the granular kidney is, that it consists primarily and essentially in a disintegration and destruction of the gland cells which line the convoluted tubes, the *débris* of the gland cells appearing in the urine as granular tube casts; that the destruction of the gland-cells induces atrophy and contraction of the tubes; that this shrinking of the tubes, with some thickening of their membranous walls and of the malpighian capsules, gives a delusive appearance of interstitial or intertubular formation of fibrous tissue; and that thickening of the walls of the arteries forms one of the most constant and conspicuous features of the disease; although this arterial change is entirely ignored by Virchow and his followers, who erroneously assume that the so-called amyloid or waxy degeneration is the only form of Bright's disease constantly and essentially associated with thickening of the blood vessels." Virchow, on the other hand, following out his classification of tubes, vessels, and interstitial tissues, divides Bright's disease into three forms, *parenchymatous nephritis*, in which the tubes are affected; *amyloid degeneration*, in which the blood vessels are implicated; and *interstitial nephritis*, where the intertubular tissue becomes thickened with subsequent atrophy and contraction of the kidney.

Cysts are not unfrequently developed in the kidney, supposed by some to be formed by the dilatation of epithelial cells; by others they are considered to be dilatations of the uriniferous tubes, which have become plugged at intervals; at any rate, cysts are more frequently found with this form of disease than with any other. The enclosed spaces then become dilated. The coats of the arteries are hypertrophied—a condition coming on towards the end of the disease. The contracted kidney is chronic from the beginning. In early life it is comparatively rare, in most cases attacking individuals after the middle period of life. It is not unfrequently associated with a gouty diathesis, and in persons thus afflicted the urine during the paroxysms of gout may be found loaded with the *débris* of cast-off cells, the urine becoming free when the gouty attack has passed off. It most frequently occurs in free livers, and those given to the excesses of the table. As one of the causes of this disease, chronic lead poisoning is given, and Dr Dickinson states that of forty-two cases of fatal lead poisoning, twenty-six suffered from granular degeneration of the kidney. The disease is sometimes associated with pregnancy. Exposure to cold, combined with intemperate habits, is probably the most frequent cause. Dropsy is absent in from a quarter to half of the number of observed cases, and when present generally consists in slight œdema of the ankles and legs, with some small amount of puffiness of the eyelids. It is most marked as the fatal termination approaches. The urine is plentiful; three or four pints a day being not unfrequently passed; the specific gravity is low, with but a small amount of albumen, but this varies with the stages of the disease. The urea, uric, and extractive matters are decreased. The urinary deposit is found to contain granular tube casts, and these, mixed with disintegrated blood cells, or granular blood casts. Sometimes hyaline casts make their appearance in the sediment.

The explanations given of the increased flow of urine in this disease are the increased blood pressure causing a greater outflow through the sound tufts; vaso-motor paralysis causing a continuous transudation through the capillaries. Dr Johnson is, however, of opinion that the copious flow of pale urine of low specific gravity is due to the secretion of a watery fluid by the tubes which have been deprived of their normal epithelium; and this opinion appears to be corroborated by the fact that the secretion is most plentiful when the denuded tubes are most numerous, that is, in the middle stages of the disease. In the earlier stages the denudation is but slight; in the latter stages the tubes have become atrophied and contracted. The arteries, as the demand for blood by the kidneys is lessened, gradually contract and become hypertrophied. The engorgement of the kidney, so marked in acute Bright's disease, is absent here; hence the comparatively small amount of albumen, and the almost entire absence of blood in the urine, so common in the former disease.

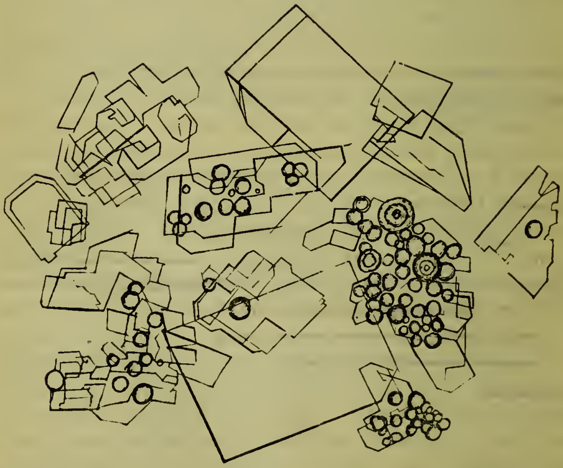
2. Large White Kidney.—In this case the kidneys become considerably enlarged, with increase of weight, due to increase in the cortical portion. The capsule peels off readily, leaving the surface beneath quite smooth. The appearance of the gland is white or whitish-yellow and anæmic, due to the obliteration of the vessels.

Total disorganisation of the kidney sooner or later takes place, and it may become the seat of fatty or amyloid degeneration. In most cases the large white kidney follows an attack of acute Bright's disease, either resulting from exposure to cold or following an attack of scarlet fever, typhus, typhoid, or diphtheria. The average age of patients suffering from this disease was, in 106 cases collected by Dickenson, 28·2 years. It is more common in males than in females. Dropsy is almost constantly present, and a marked tendency to secondary inflammations has been noticed. Uræmia is also not unfrequent. The urine is scanty, the specific gravity at one time nominal, at another time slightly raised—the colour may be pale and cloudy, or smoky,—and deposits tube casts, epithelial cells, etc. The disease in most cases has a protracted course, but death may

Chronic Desquamative or Tubal Nephritis
Parenchymatous Nephritis

take place in about six months. When life has been prolonged for some time the kidneys may become contracted and granular, but this condition must not be mistaken for the granular contracted kidney.

3. Fatty Kidney.—In this form of the disease the renal structure becomes soft, yielding, and of a fawn colour, sometimes here and there presenting small red vascular patches. The kidneys are also more or less enlarged. When cut into they present a fatty appearance, and give a greasy, unctuous sensation to the finger. The usual gland cells are uniformly infiltrated with oil. This condition of the kidney is most frequently found in those who have suffered from diabetes or other wasting disease. The blood becomes fatty, the fat being arrested in the secreting cells of the kidney. It also occurs along with fatty degeneration in other organs.



Cholesterine obtained from the fatty matter in casts separated from the urine of a case of fatty degeneration *(Beal).*

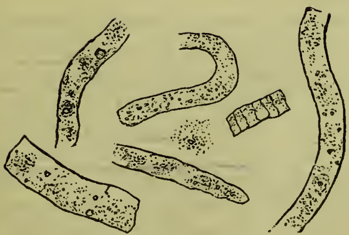
4. Lardaceous, Amyloid, or Waxy Kidney.—This condition is rare, but in most cases occurs in strumous, syphilitic, or phthisical patients. The organs may or may not be increased in size. They are found dense and firm, and present a waxy appearance. The renal structure gives one the idea of its having been soaked in balsam. Lardaceous or waxy kidney is generally accompanied with a like condition of the liver or spleen. The natural structure of the gland is almost entirely lost; it is hard, not easily torn, and the capsule can generally be removed with ease. The disease, according to Virchow, has its origin in the arteries and malpighian capillaries; according to Johnson, in the secreting cells, the vessels becoming secondarily affected. A watery solution of iodine gives a dark mahogany brown colour with that portion of the kidney which is diseased, the healthy portion only giving a yellowish tinge. The lardaceous material is not of the nature of starch or cellulose, as supposed by Virchow, but more probably fibrin more or less modified. In the earlier stages the urine is generally abundant, pale in colour, and of low specific gravity. The abundant flow—50 to 100 ounces daily—of pale urine without sugar may, till it becomes albuminous, be mistaken for a case of diabetes insipidus. Tube casts are rarely present in the early stages. Dropsy most frequently occurs, but uræmic poisoning is of rare occurrence. The albumen gradually increases as the disease proceeds; the urine becoming scanty, pale in colour, and the specific gravity high. The patient looks pale, sallow, and cachectic.

These several affections cause—

1. *Interference with the secretory functions of the kidneys*—
    - (*a*) By plugging the convoluted tubes with disintegrated epithelium.
    - (*b*) Destruction of the secreting structure.
2. *Interference with the circulation in the kidneys*—
    - (*a*) Congestion takes place.

3. *Poisonous effects due to*—
    - (*a*) Uræmia, attributable to the non-elimination of urea.
    - (*b*) Albuminuria—to be attributed to interference with the circulation through the kidney.

General Symptoms.—In many cases the first symptom that attracts the attention of the patient to his kidneys is the frequent desire to make water, especially at *night.* In other cases, however, the appearance of dropsy is the first indication of renal mischief. This tendency to frequent micturition *during the night* in incipient Bright's disease is important, especially if the patient be young, and there be no suspicion of prostatic disease. Frequent micturition, due to the presence of calculus, more often occurs *during the day,* caused by the irritating effect of the movement of the stone in the bladder. Premonitory to the above, however, the patient may, for some time previous, have complained of *dyspepsia,* with general and increasing debility, emaciation, accompanied with marked pallor of the face. Pain in the back is as frequently as not absent, and it must be borne in mind that extensive disease of the kidney may have gone on for some time without attention being directed to the kidneys. The dropsy, a marked symptom in the large white kidney, usually makes its appearance first as an œdematous swelling round the eyelids, most marked in the morning when the patient rises from his bed. This condition may also occur simultaneously with anasarca of the lower extremities, scrotum, and penis. If the urine be now examined it will be found of low specific gravity, pale in colour, and containing albumen in considerable quantity. The quantity passed varies with the condition of the kidney. In the contracted form of the disease, its quantity may not be very materially diminished, and the amount of albumen not large, sometimes disappearing for a time. In this form of the disease, dropsy is *not* a prominent symptom. On the other hand, the urine passed when the disease takes the form of the white kidney is scanty, full of albumen, and of low specific gravity, a great diminution in the amount of the solids having taken place. The urea can be detected in the blood. Sometimes in the early stage, when the acute disorder is passing off, the urine assumes a dark smoky hue, due to the presence of blood changed in colour by the action of the acid urine. A more hopeful prognosis may be given when the urine is in this condition than when it assumes a pale straw colour. If some of the urine be placed in a test tube and heat be applied, the albumen will be precipitated. A few drops of acetic acid should be added. This precaution is rendered necessary by the fact that in weakly persons heat alone may produce a white deposit of the phosphates, not unlike the albuminous deposit, but which on the addition of a few drops of acetic acid is dissolved. Sometimes, also, if the urine be alkaline, heat alone will not cause a coagulation of the albumen. Nitric acid added to fresh urine may precipitate the uric acid, giving a cloudy appearance. Both heat and nitric acid should therefore be used to save the possibility of error. Examined by the microscope, the urine may be found to contain *granular casts*, *fatty casts*, *hyaline* or *waxy casts*, and renal epithelium cells, oil drops and granular matter.



Granular casts from the urine of a patient suffering from *chronic inflammation* of the kidney *(Beal)*.

The early symptoms now become more marked. The continued paleness of the face points to an anæmic condition of the system. The digestive organs now show more prominent symptoms of general disorder, and there may be present frequent attacks of sudden vomiting. The respiration and the circulation become more or less impeded from the dropsical effusion in the pleura, pulmonary œdema, and œdema of the glottis, together with hydro-pericardium, etc. Pericarditis and endocarditis may also occur, and may be detected by the physical signs common to those affections.

The small contracted kidney is not unfrequently associated with a gouty diathesis. Hypertrophy of the left ventricle without valvular disease may occur during the course of Bright's disease. The cause for this, which occurs most frequently in cases of contracted kidney, is not very evident. Dr Johnson is of opinion that the circulation of blood containing urinary excreta causes a contraction of the small arteries of the body, requiring increased action of the left ventricle to propel the blood. A full resisting pulse, marking high arterial tension, with the absence of valvular disease, but with the second sound of the heart heard more distinctly over the aortic valves, and displacement of the apex, points to chronic and advanced renal disease. A doubling of the first sound of the heart is another sign of this arterial tension.

The amount of albumen passed in twenty-four hours may vary from forty to five hundred grains. The urine is usually pale in colour, cloudy, and may contain blood in varying proportions, the quantity, however, being in most cases very small. The urea, together with the other solid constituents of the urine, are all diminished. The blood becomes more watery, the red corpuscles less in number, and the quantity of albumen is below the normal standard. Hæmorrhage from the nose, often very difficult to arrest, is not unfrequent. Intercranial hæmorrhage is by no means an uncommon occurrence, due to the diseased condition of the arteries generally. The skin is frequently dry and harsh to the touch. It is with difficulty made to act even under the influence of the hot-air bath. By some this inactivity of the sweat-glands is held to be due to contraction of the arteries of the skin; by others, to disease of the glands, resulting from the unhealthy condition of the blood.

The blindness, or rather a dimness of vision, sometimes present in the course of chronic Bright's disease, is interesting, and is important as an aid to diagnosis. The attacks in some cases are, as a rule, sudden, passing off rapidly, but occurring again and again. In other cases the impairment of vision is more slow and more lasting. In the one case, when the eye is examined by the ophthalmoscope, no structural changes are apparent; in the other, the structural changes present are an effusion into the choroid and retinal coats of the eye, and gradual and progressive atrophic changes.

A distressing form of dyspnœa, "renal asthma," coming on at irregular and uncertain times, chiefly at night or after food, in the course of chronic Bright's disease, is the result, in all probability, of a sudden and powerful contraction of the pulmonary arterioles. Hertz and Rosenstein, however, consider this renal asthma to be due to œdema of the bronchial mucous membrane. On auscultation, loud sibilant *râles* may be heard over the lungs, or loud puerile respiration, unaccompanied with *râles* or crepitation. On the whole, however, there are no physical signs adequate to explain the distressing sensation of "want of breath." This complication may probably be one of the results of uræmic poisoning.

Causation.—The causes which give rise to chronic Bright's disease are obscure. It may follow the acute affection. Gouty or strumous persons are often the subjects of this disease, as are also those who have suffered from syphilis, and workers in lead.

Diagnosis.—The presence of dropsy, together with the concurrent appearance of albumen and tube-casts in the urine, will in most cases assist in pointing out the nature of the disease from which the patient suffers.

The *Prognosis* is always unfavourable.

Treatment.—A knowledge of the previous history of the patient; presence or absence of an acute attack, gout, syphilis, intemperance, hereditary taint, are all-important in the treatment of this disease, which will consist in supporting the strength of the patient, and in treating the complications as they arise. The patient should be warmly clad, flannel being worn next the skin. An easily digested and nourishing diet will be required. Alcohol should be sparingly used, if given at all. The preparations of iron are indicated, especially when there is a marked anæmic condition of the blood. The dropsy is best treated by purgatives, the most useful being the compound jalap powder, combined with the bi-tartrate of potash. In severe cases elaterium may be given, together with diuretics, their effects being carefully watched. Flannels steeped in a strong infusion of digitalis may be applied over the abdomen. Turkish baths are sometimes useful to cause a free action of the skin, and, where these fail, the "wet pack" may be tried. The lower extremities may be punctured with a lancet. This often gives intense pain, and I have never seen any permanent good derived from it. Mercury, in no form, should be given in Bright's disease, as it often acts most injuriously. Renal asthma may be relieved by the administration of hydrate of chloral and the bromide of potassium. If the stomach be too irritable, the chloral may be injected into the rectum. Opium is contra-indicated in most cases of this disease. Uræmic symptoms, when they occur, will require the use of active purgatives, the hot-air bath, and diuretics to remove the morbid materials from the blood.

TABLE GIVING THE FORMS OF CHRONIC BRIGHT'S DISEASE.

Synonyms—	Chronic Desquamative or Tubal Nephritis. 'Large White Kidney.' Parenchymatous Nephritis.	Amyloid, or 'Lardaceous Kidney.'	Interstitial Nephritis. Contracted, Granular, or 'Cirrhotic Kidney.'
Cause.	Most frequently a sequel of the Acute Kidney "Scarlatina."	Suppuration.	Drink, Gout, Lead.
Dropsy.	Very great early.	Great towards termination of disease.	Slight at first; afterwards more marked. Sometimes absent.
Urine—Quantity.	Diminished.	Increased.	Much increased.
" Sp. Gravity.	About Normal.	1005 to 1015.	1002 to 1010.
" Albumen.	Abundant, Smoky (?)	Abundant, increasing with progress of attack.	Small in quantity. Sometimes absent.
" Casts.	Blood, Epithelial, Granular, Hyaline, Fatty.	Hyaline, if any, but very few usually.	Hyaline, Granular, and Fatty.
Uræmia.	At any period, but sometimes even absent.	Very rare.	Usually late in appearance, if at all.
Complications.	Pneumonia, Peritonitis, Œdema of Lungs. Blindness.	Enlarged Liver, Enlarged Spleen.	Hypertrophy of Left Ventricle, Apoplexy, Cirrhosis of Liver, Retinal Hemorrhages.

SYNOPSIS OF SYMPTOMS AND POINTS OF DIFFERENCE BETWEEN 'LARGE WHITE KIDNEY,' 'CONTRACTED KIDNEY,' AND 'LARDACEOUS OR WAXY KIDNEY.'

LARGE WHITE KIDNEY.	CONTRACTED KIDNEY.	LARDACEOUS OR WAXY KIDNEY.
1. Generally follows acute Bright's disease, scarlet fever, or repeated pregnancy.	1. Chronic from the beginning.	1. Generally comes on insidiously in cachectic persons suffering from debility.
2. Dropsy is frequently present, and the body is œdematous.	2. Dropsy, in the majority of cases, is absent, or limited to slight œdema of the eyelids.	2. Dropsy, in the majority of cases, is present.
3. Great tendency to secondary inflammations, pneumonia, and peritonitis, and œdema of the lungs. Uræmia not unfrequent.	3. Strong tendency to hypertrophy of the left ventricle, atheroma of the arteries of the brain, apoplexy, and uræmia.	3. Uræmia seldom occurs. Generally complicated with *phthisis*, *syphilis*, and *waxy* degeneration of *liver* and *spleen*.
4. The urine is scanty, sp. gr. normal or slightly raised, pale and cloudy, sometimes 'smoky.'	4. Urine plentiful, and of low specific gravity. Towards the latter stages urine may become scanty.	4. The urine is abundant in quantity, pale in colour, and albuminous.
5. The deposit is composed of 'fatty,' 'hyaline,' 'epithelial,' and 'granular' casts.	5. The deposit, slight in quantity, is composed of granular and hyaline casts, some epithelium, seldom fat or blood.	5. The deposit is scanty, consisting in atrophied renal cells.
6. Albumen is always present.	6. The quantity of albumen is small, and may disappear for a time from the urine.	6. Albumen is always present.
....	7. Constant desire to pass water.	....
....	8. Impairment in vision, coming on suddenly, and then passing off or remaining for some time.	....

URÆMIA.

Definition.—Uræmia or uræmic poisoning are terms employed to denote a certain condition of the nervous system, produced by the circulation of effete matters in the blood, and characterised by epileptiform convulsions, ending in most cases in coma.

The nervous phenomena of uræmia not unfrequently occur during the course of both the acute and chronic forms of Bright's disease of the kidneys, or when from any other cause the action of the kidneys is impeded or arrested. The most prominent symptoms are headache, and vomiting, perhaps suddenly ceasing, to give place to a feeling of heaviness and a desire to sleep. This state may pass off, or be followed by epileptiform convulsions, coma sooner or later supervening, and terminating in death. Consciousness, in rare cases, is not entirely lost.

In all cases which admit of a doubt as to the nature of the case, the urine should be carefully examined, some being withdrawn, if necessary, by means of a catheter, for that purpose. Immediately preceding an uræmic attack, the urine in most cases becomes diminished in quantity, together with a like diminution in the amount of urea. But it must be borne in mind that total suppression of urine may occur, and may continue for some days without the supervention of uræmic symptoms. See the record of an important case in the *Medical Examiner*, 15th November 1877, in which no urine had been voided for 9¼ days, owing to both ureters being plugged by calculus. Diarrhœa and vomiting are not uncommon in Bright's disease, and may to a great extent ward off the uræmic attack by getting rid of some of the poison in the blood.

The question now arises as to causation in the absence of any anatomical lesion in the brain to explain the peculiar phenomena noticed in uræmia. Richardson and Hammond consider that urea is the active agent. Frerichs believes that the urea is harmless, and that it is its conversion into carbonate of ammonia in the blood which induces the nervous symptoms. Treitz maintains that this conversion of urea into ammonia is not carried on in the blood-vessels, but that it is first excreted into the alimentary canal, where it is converted into the carbonate of ammonia by the gastro-intestinal mucus. Uræmic diarrhœa and vomiting may be thus explained.

As far as the above attempted explanations go, it may be stated that Hammond and Richardson have proved that ammonia exists in the blood in healthy animals. Dr Johnson is of opinion that uræmic convulsions are the result of anæmia of the brain, due to the contraction of the vessels from the irritating effect of the poisoned blood circulating through them. Oppler, Schottin, and Perls state—and they believe themselves warranted in doing so from the nature of their experiments,—that these uræmic nervous phenomena are due to the accumulation in the blood of the primary products of tissue metamorphosis, which are on their way to be transformed by the kidneys into urea and uric acid. The proof of this, they assert, is to be found in the fact that urea accumulates more abundantly in the blood of animals in whom they had tied the ureters, than in those from whom they had removed the kidneys. They also state that the blood contained the same amount of urea as in health in animals who had lost their kidneys, but that this substance became increased when the ureters were tied, and that an increase of ammonia in the blood did not follow the presence of large quantities of urea. *Creatin* was, however, always found increased in the muscles of the animals.

Uræmic convulsions do not, as a rule, follow simple obstructive suppression of the urine.

Treatment.—This will consist in the administration of purgatives, diaphoretics, and diuretics.

THE FOLLOWING TABLE WILL ASSIST THE STUDENT IN FORMING A DIAGNOSIS :—

URÆMIC COMA.	APOPLEXY.	OPIUM POISONING.	EPILEPSY.
The absence of paralysis. Return of consciousness during the convulsive attacks, if any are present. The pupils are dilated, or semi-dilated, and there are remissions in the insensibility. Face pale, breathing easy.	Paralysis. The age of the patient, and previous history, will assist in forming a diagnosis.	The pupils contracted. No remissions in the insensibility.	Diagnosis difficult. The history of the case will help, and also an examination of the urine. Face flushed and purplish.

TABLE SHOWING THE CONDITION OF THE URINE IN HEALTH AND IN CERTAIN DISEASES :—

HEALTH.—Quantity—About 50 ounces of urine are passed by a healthy adult in twenty-four hours; children relatively more than adults; males more than females; exercise, causing cutaneous and pulmonary exhalation, diminishes the daily quantity. Sp. gr. 1·020. Colour, that of amber; reaction as a rule acid, portions collected at different times being sometimes neutral, at others alkaline; but the acidity and alkalinity varies, forming the *acid* or *alkaline tides* in the twenty-four hours—the acid tide during the night and before meals, the alkaline after meals, hence during the day. The urea varies with the diet, greater when nitrogenous food is taken; uric acid small, chlorides variable, odour not unpleasant, but affected by articles of diet, asparagus, medicines, rhubarb, turpentine, causing odour of violets, etc.

AGUE.—During the hot and cold stages the water of the urine is increased in quantity, but during the sweating stage, the amount is diminished. During the fit the urea is increased; this is also the case with the uric acid. The chloride of sodium is greatly augmented in quantity during the cold and hot stages.

ANÆMIA.—The urine is pale, there being a marked deficiency in the amount of pigment.

CHLOROSIS.—It is nearly neutral in reaction, and of low specific gravity.

CHOREA.—The specific gravity varies from 1019 to 1035. Lithate of ammonia and oxalate of lime are frequently present. The urea is also in excess. The density to a great extent depends upon the excessive movements of the muscles in patients suffering from chorea.

DIABETES.—The odour is sweetish or like hay. The colour is a pale straw colour. The specific gravity varies from 1030 to 1074. If allowed to stand for any time it ferments.

DYSPEPSIA.—The urine sometimes contains free acid. Cardialgia, nausea, and frontal headache are frequently associated with an acid condition of the urine soon after food has been taken. A diminution of the chloride of sodium is most marked when digestion has been imperfect. Crystals of oxalate of lime are common in this disease.

JAUNDICE.—The urine is of a saffron, dark-olive green, or nearly black colour. Bile pigment, leucine, and tyrosine are present.

PNEUMONIA.—During the first stages the watery portion is lessened. The urea is increased in quantity. Specific gravity 1025 to 1035. Uric acid and the colouring matter are also greater than in health. The chlorides are diminished when hepatisation commences, and reappear with the stage of resolution. The sulphuric acid is increased to about one-third the nominal amount, and albumen may or may not be present.

TYPHOID FEVER.—Water diminished in quantity.
Specific gravity is high.
The urea and uric acid are increased.
Chloride of sodium less than normal.
Sulphuric and phosphoric acids, normal.
The colouring matter is sometimes increased.
Albumen and tube casts may be sometimes present.

TABLE SHOWING THE EFFECTS OF DISEASE ON CERTAIN CONSTITUENTS OF THE URINE:—

	INCREASED.	DIMINISHED.
Quantity.	Hypertrophy of left ventricle, early stages of chronic Bright's disease, hysteria, diabetes mellitus, and insipidus. After large draughts of water.	Shock, febrile diseases, diarrhœa, acute inflammation of the kidneys, last stages of chronic Bright's disease. Exercise.
Specific Gravity.	Diabetes mellitus, full meat diet.	Large draughts of water. Hysteria.
Urea.	In health depends on nitrogenous food taken; febrile diseases on rapid tissue disintegration; chronic pulmonary disease (?); diabetes (?)	All chronic diseases without fever. Cancer, cirrhosis, and abscess of the liver. Cancer. Bright's disease.
Uric Acid.	In fever, functional and organic disease of liver, disease of spleen, leucocythæmia, and diseases of lungs preventing elimination of CO_2.	
Chlorides.	In pneumonia as soon as resolution commences. Ague in cold and hot stages.	In all febrile diseases. In pneumonia during hepatisation. Acute rheumatism with joint effusion, in pleurisy with effusion.
Phosphates.	In many chronic diseases with tissue waste, diabetes.	
Sulphates.	Preponderance of animal food.	In prolonged abstinence from food.

Table of the Examination of the Urine[*] and Tests for following Substances:—

Chlorides.—1. Collect urine for 24 hours. 2. Take 50 c.c. of urine, dilute to 100 c.c., neutralise with sodic carbonate. 3. Slightly tint with solution of potassium chromate. 4. Gradually run in from a burette small quantities of solution of argentic nitrate, stirring after each addition. 5. Stop the process when a permanent red colour is produced in the urine.

Silver Solution, 20·063 grm.; distilled water, 1 litre.

Rule.—1 c.c. silver solution equals ·006 grm. HCl or ·01 grm. NaCl. 10 c.c. of silver solution used, hence $\cdot006 \times 10 = 0\cdot060$. Total urine passed, 1800 c.c. in 24 hours, hence $\dfrac{0\cdot060 \times 1800}{50} = 2\cdot16$ grm. HCl, or $\cdot01 \times 10 = 0\cdot10$ then $\dfrac{0\cdot10 \times 1800}{50} = 3\cdot6$ grm. NaCl.

To determine chlorine proceed as follows:—

$$\begin{array}{ll} \text{Na} = 22\cdot99 & 58\cdot49 : 3\cdot6 :: 35\cdot5 : x \\ \text{Cl} \quad \underline{35\cdot5} & x = 2\cdot1 \text{ grm. Cl} \\ \quad\;\; 58\cdot49 & \end{array}$$

Albumen.—1. Take an inch and a-half of urine in a test tube. 2. Add a drop or two of acetic acid and heat. 3. A deposit takes place; albumen (?) or earthy phosphates (?). 4. Add nitric acid, phosphates disappear, albumen permanent.

Precautions.—If amount of albumen be small, an excess of nitric acid may hold it in solution, hence acidify with acetic acid, and then add the nitric. Turpentine and copaiba are precipitated by nitric acid but dissolved by alcohol. Heat also dispels the cloud formed by copaiba.

Bile Pigments.—*Gmelm's Nitrous Acid Test.*—1. Take an inch and a-half of urine in a test tube. 2. Pass slowly down the side of the test tube a little fuming nitric acid. 3. At the union of the two a play of colours, green, blue, violet-red and yellow, or yellowish green. 4. The colours are from above downwards, the highest colour being the important one.

---

[*] For further information, see the Author's book on "The Urine."

Heller's Test.—1. Take about one drachm of HCl. 2. Add slowly enough urine to colour it. 3. Mix. 4. Pour slowly down test tube pure nitric acid to "underlay" urine. 5. Play of colours at union of two liquids.

Fleischl's Test.—1. Add pure nitric acid to urine. 2. Mix. 3. Pour slowly down test tube concentrated sulphuric acid to "underlay" the other liquid. 4. Play of colours at junction of the two liquids.

SUGAR.—For Tests. (See p. 161.)

BLOOD.—Examine for blood cells under microscope.

Guaiacum Test.—Add to the urine in a test tube a few drops of tincture of guaiacum and then some ozonised ether (ethereal solution of *peroxide of hydrogen*) and shake, when a blue colour is formed on the surface of the urine.

Precautions.—Saliva, a salt of potassium, and nasal mucus all strike a blue colour with guaiacum, even with the addition of ozonic ether; albumen in any quantity interferes with the test.

Hœmatin Test.—1. Precipitate earthy phosphates from urine placed in a test tube with caustic potash, heat gently. 2. Filter and dry phosphates on a slide. 3. Add a crystal of common salt and cover with a cover glass. 4. Put a drop of glacial acetic acid on slide and allow it to come in contact with the phosphates and salt. 5. Warm the slide. 6. Allow to cool, and examine for hæmin crystals under microscope.

DIABETES INSIPIDUS.

Lat. *Diabetes insipidus.* Fr. *Diabète non sucré.*
Ger. *Diabetes insipidus.* Syn. *(Die) zuckerlose Harnruhr.*

Chronic diuresis or polyuria are terms applied to an affection characterised by an increased secretion of urine of low specific gravity, and accompanied with intense thirst. The disease occurs more frequently among men than women. It has been attributed to cerebral disease, blows on the head, intemperance, exposure to cold, certain inflammatory diseases, and to hereditary predisposition.

The invasion of the disease is frequently sudden, no premonitory symptoms preceding the attack. The skin is usually dry and harsh. The thirst is intense. The general health of the sufferer is in many cases not much impaired.

Diabetes insipidus must not be confounded with diabetes mellitus, in which latter disease, besides the great increase in the quantity of the urine, there is the well-marked and persistent presence of sugar.

The exact nature and pathology of this disease has not yet been clearly defined. The primary cause, it would appear, must be looked for in other parts than the kidneys. Bernard found that by irritating the floor of the fourth ventricle of the brain, he could produce an increased flow of urine containing neither albumen nor sugar. The sympathetic nervous system is probably more or less implicated in the production of this disease. There is no condition of the kidney peculiar to this disease.

HÆMATINURIA.

Definition.—A disease of the blood in which the blood discs become destroyed, and the colouring matter or hæmatine is alone discharged in the urine, accompanied with the presence of albuminuria. The renal capillaries are not ruptured.

Hæmatinuria must be distinguished from hæmaturia, in which latter disease the blood discs may be detected, with the aid of the microscope, in the urine. Hæmatinuria may assume a paroxysmal or intermittent type, the paroxysm being ushered in with a sensation of chilliness. In some cases the attack resembles the early stages of ague. The urine passed soon after the shivering fit is more or less of a red, bloody character, but, as a rule, regaining its normal colour on the second or third act of micturition after that containing the colouring matter of the blood. Previous to the acute stage the patient complains of general malaise. The urine may resemble porter, from the amount of blood-colouring matter present. The specific gravity varies from 1015 to 1033. The disease may return at varying intervals for several years, the prognosis on the whole being favourable. Males are, as a rule, attacked, but the cause and pathology are alike obscure. Exposure to cold, or malaria, has accounted for one or two cases; but the disease seems to be the result of a sudden congestion of the real capillaries, how brought about it is not easy to say.

Treatment.—Treat by cupping, and vapour baths to the loins. Tannic or gallic acid may be tried, and iron and quinine also given, especially the latter if there is any history of ague. Rest in bed during the paroxysm should be enjoined.

HÆMATURIA.

Lat. *Hæmaturia renalis.* Fr. *Hématurie.* Ger. *Blutharnen.*

Definition.—Blood mixed with the urine, and more or less diffused through that secretion, giving to it a reddish or smoky appearance.

This disease may be due to several causes; thus it may result from injury or sudden congestion of the kidneys. It may be symptomatic of purpura, scurvy, small-pox, and the other eruptive fevers, also in cholera, ague, etc. It may take the place of normal menstruation, or the bleeding from hæmorrhoids, etc. Hæmaturia may follow severe mental emotion.

When blood is found in the urine, the question may arise as to the locality whence it comes. If diffused through the urine, giving to it a smoky tint, the blood is from the kidneys; if, on the other hand, the colour is pinkish or reddish, the source of the hæmorrhage is most probably the urethra, bladder, or ureters. The passive hæmaturia occurring in any of the zymotic diseases must be distinguished from acute Bright's disease.

Treatment.—The cause must be sought and considered before any line of treatment is followed. When due to injury, rest and cold applications to the loins. Styptics, acetate of lead and opium, preparations of iron, gallic or tannic acids. Ergot, especially the subdermic injection of ergotin, is of much use in severe cases. In vicarious hæmaturia, as in cases of suppression of the menses and constipation, purgatives and iron, F. 9 and F. 26, must be given. If the bladder is suspected to be full of blood, use a large-eyed catheter and injections of cold water with 20-40 grains of alum to each pint of water, as recommended by Dr Prout.

CHYLURIA.

Definition.—A disease chiefly of tropical countries, and characterised by the discharge of a milky coloured urine; sometimes, however, of a pale salmon pink colour from admixture with blood. The disease is supposed to be due to the presence in the blood of a parasite, the Falaria Sanguinis Hominis, which, puncturing the lymphatics, allows of the escape of their contents.

The invasion of chyluria is most variable; a sudden discharge of chylous or lymphous urine, attributed to some late accident or mental emotion, being, in many cases, the first evidence of the presence of the disease. The characteristic condition of the urine may occur at irregular intervals without any premonitory warning. For a long time this curious disease baffled all attempts made to account for its presence, but one point, not clearly defined, seemed to suggest some connection between residence in the tropics and the occurrence of the disease. Later researches, have, however, gone far to show that the presence of a parasite nearly allied to the *Bilharzia hæmatobia*, to which the paroxysmal hæmaturia of tropical countries is attributed, may be the cause of chyluria. This parasite is known as the *filaria sanguinis hominis*. For some interesting information on the subject, see *British Medical Journal*, October 22, 1881, p. 668. It now appears that the mosquito has not a little to say about the pathology of of the disease, and that the filaria swarm in the human subject just at the time when the female mosquito visits the body of its victim, and it is suggested that the mosquito is an intermediate host.

The urine in this disease, as before said, is milky, becoming on standing a trembling jelly-like mass, which, however, sooner or later breaks up into flaky clots.

Treatment.—The treatment at present consists in the administration of iron and gallic acid.

RENAL COLIC.

This is due to the passage of a calculus from the kidney along the ureter to the bladder, or by the irritation caused by a concretion in the pelvis of the kidney.

Symptoms.—These frequently set in with marked suddenness, the patient complaining of the most acute pain shooting from the kidney towards the bladder, but the pain may spread all over the lower part of the abdomen. The patient may be covered by profuse perspiration due to the intensity of his suffering. Micturition may be frequently accompanied with pain along the urethera and retraction of the testicle. The patient may place himself in a variety of postures in the vain attempt to relieve his suffering, but, as a rule, is found on his back with his knees drawn up towards his abdomen. The pain may as suddenly cease, due to the escape of the calculus into the bladder, or from some other cause, as the cessation of pain is no proof that the stone has entered the bladder. Irritation of the renal pelvis by a concretion may give rise to renal colic, so also may the presence of blood or echinococcus in the ureter. The stretching of the ureter by an abnominal aneurism has been known to give rise to renal colic.

Treatment. — Opium, chloroform, etc., to relieve pain. Dilutent drinks in the hope of washing the calculus into the bladder. Hot baths are sometimes of use. If uric acid calculi be suspected large doses of soda or liquor potassæ freely diluted may be given to dissolve the calculus. If these means fail in relieving the patient recourse must be had to operation, *nephro-lithotomy*, as it has been called.

CANCER OF THE KIDNEY.

The kidneys, though rarely, may become the seat of cancer, in which case it is usually the encephaloid variety. Drs Wilks and Moxon are of opinion that in most of the so-called cases of cancer of the kidney the disease has arisen primarily in the lymphatic glands or other parts outside the kidney, chiefly about its hilus, the organ itself being comparatively untouched. In some cases, however, the entire structure of the gland may be infiltrated and the kidney enlarged.

MOVABLE KIDNEY.

Lat. *Renes mobiles.* Fr. *Rein mobile.* Ger. *Abnorme Beweglichkeit der Niere.*

This disease is due to some obscure cause. One of the kidneys—in most cases the right—may become loosened, and so form a movable tumour distinctly felt through the abdominal walls. This anomalous condition of the kidney gives rise to dull aching pain in the lumbar region, increased by pressure or muscular exertion. It is well to remember this fact when about to determine the nature of any obscure abdominal tumour.

The use of an abdominal belt will in a measure obviate any inconvenience that may attend this condition of the kidney.

HYDRONEPHROSIS.

Lat. *Hydrops renum.* Fr. *Kystes.* Ger. *Hydronephrose.*

Definition.—A dilitation of the pelvis and glanular substance of the kidney into one or more cysts by retained secretion.

Hydronephrosis is a condition of the kidney attended with more or less complete destruction of the gland, by the accumulation of fluid in it. The collection of fluid may depend upon congenital malformation of the kidney, or it may be due to some obstruction—impaction of renal calculus in the ureter, by which the secreted urine is prevented from passing off through its natural channel. One or both kidneys may become affected.

The term "dropsy of the kidney" has been given to this condition, but it is by no means a good one. The fluid is, in most cases, urine more or less modified.

It is important in the examination of the abdomen, with regard to doubtful tumours, to bear in mind the possibility of this state of the kidney. The enlarged kidney has been tapped, under the impression that it was a case of ovarian dropsy. When not due to congenital malformation, the obstruction in the ureter may be removed, and a sudden flow of urine mark the patency of the duct.

THE FOLLOWING TABLE MAY ASSIST IN FORMING A DIAGNOSIS, WHICH AT THE BEST OF TIMES IS NOT EASY:—

OVARIAN DROPSY.	HYDRONEPHROSIS.	ASCITES.	HYDATID CYSTS.
Abdomen more enlarged on one side than on the other. Dulness due to the cellular condition of ovary, and its presence *before* the intestines.	A tympanitic sound heard in front, due to the presence of the colon in front of the tumour, dulness in the lumbar region. Dulness is fixed in whatever position the patient may be placed.	Dulness on percussion will depend upon the position of the patient. If lying on the back, dulness at back, and *vice versa*.	Escape of hydatid cysts with urine, or presence of hydatid fremitus. Hydatid cysts of the kidney are seldom found on both sides.

N.B.—In all doubtful tumours in the abdomen, pass a catheter; you will thus get rid of one source of error—*distended bladder*.

Table showing the several Forms of Gravel, the Symptoms which accompany their Formation and Presence in the Urine; their Treatment, and the Tests by which they may be Distinguished.

	LITHATES.	PHOSPHATES.	OXALATES.
Cause	Peculiar condition of the system. Errors in diet, sedentary habits. Dyspepsia, gout, and rheumatism, and hereditary predisposition.	Exposure to cold, injury to spinal cord. Catarrh of the bladder, any irritation of the bladder. Peculiar condition of system.	A constitutional predisposition; the use of certain vegetables — carrots, turnips, etc.; impediment in the respiratory functions; during convalescence from severe diseases.
Symptoms	Irritation felt in the urethra when passing water. Pain felt shooting down from kidney into the groin or bladder. The pain intermits. Nausea and vomiting are not unfrequent. The pulse is not increased in rapidity, and the temperature of the surface of the body is normal. The urine deposits a pink, yellow, or red sand.	Pain felt in the back and loins. Digestion imperfect. Bladder irritable. Urine contains mucus, and may contain, when passed, a white milky deposit. Marked tendency to crystallise round any nucleus, hardened mucus, etc. Urine alkaline from volatile alkali betokens disease of urinary organs.	No well-marked symptoms. Patient's attention first attracted by pain on passage of calculus from kidney.

Table—*Continued.*

TABLE SHOWING THE SEVERAL FORMS OF GRAVEL, ETC.—*Continued.*

	LITHATES.	PHOSPHATES.	OXALATES.
Diagnosis......	The red deposit, when abundant, may be confounded with blood. Absence of mucus. The urine is acid, and does not become alkaline or decomposed on standing. The lithates, when precipitated, are dissolved by heat; the phosphates are not.	The urine, at first acid, becomes alkaline and putrescent on standing. Ammonia throws down a white cloud of phosphate of lime and ammonio-magnesian phosphate. The addition of phosphoric acid re-dissolves the precipitate.	Presence of a sediment which is not soluble in acetic acid; soluble in hydrochloric acid, and is then precipitated by ammonia.
Prognosis......	Favourable, if the deposit is slight. Danger will be increased if a calculus is formed.	Danger will depend on concurrent vesical disease, and the formation of a calculus.	Formation of calculus of the mulberry variety.
Treatment—Medicinal....	Administration of the alkalies, especially the bicarbonate of potash. The salts of lithia, colchicum wine, and turpentine are sometimes of use.	Tinctures of bark and iron, and the mineral acids, especially the nitro-hydrochloric, are the medicines generally used.	The administration of the nitro-hydrochloric acid is often of use.

TABLE SHOWING THE SEVERAL FORMS OF GRAVEL, ETC.—*Continued.*

	LITHATES.	PHOSPHATES.	OXALATES.
Dietetic..........	A vegetable diet; less animal food. Avoidance of alcohol. Barley water, etc.	Generous diet, including meat and small quantities of wine.	Avoidance of all vegetables which contain oxalic acid—rhubarb, carrots, sorrel, etc. — and sparkling wine or beer.
Tests.............	Urate of ammonia insoluble in cold, but soluble in hot urine. The urine heated and filtered will deposit urate of ammonia on cooling. The urine is acid. Uric acid is insoluble in cold or hot urine, in acetic or hydrochloric acids.	Earthy phosphates are not dissolved by heat, but they are soluble in acetic acid. The urine acid at first becomes alkaline, and putrefies on standing.	Oxalate of lime is insoluble in acetic acid, but soluble in hydrochloric acid, from which it is precipitated by ammonia. Insoluble in phosphoric acid.

DISEASE OF THE SUPRA-RENAL CAPSULES.

ADDISON'S DISEASE.

Lat. *Morbus Addisoni.* Fr. *Maladie d'Addison.*
Ger. *Addison'sche Krankheit.*

DEFINITION.—Disease of the supra-renal capsules, with discoloration of the skin.

Disease of the supra-renal capsules appears to be sometimes attended with a peculiar form of anæmia, in which a discoloration of the skin forms a prominent feature.

This disease, generally known as *Addison's* or the *bronze-skinned disease*, is probably the result of a tuberculous inflammation of the supra-renal capsules, and is in most cases associated with phthisis. The capsules are enlarged, hardened, and when cut into may be found more or less of a cheesy consistence. The pathology of this condition has still to be studied before any decided opinion can be given as to the mutual relationship between the peculiar colour of the skin and the disease of the capsules. One thing is certain, namely, that the capsules may be entirely destroyed by cancer or lardaceous change, and yet the symptoms peculiar to Addison's disease be absent. Some are inclined to attribute the disease to the slow chronic and complete nature of the destruction of the capsules, and point to the fact that the peculiar coloration of the skin is also produced by other tuberculous diseases. Others refer to the highly nervous composition of these organs, and attempt to explain the disease in question by pointing out the nervous connection between them and the solar plexus. By the complete disintegration of the capsules, the abdominal sympathetic nerves are affected, which, according to Risel, causes a determination of blood to the abdominal organs, at the same time injuring their functions, and thus producing the anæmic condition, one of the chief signs of Addison's disease. In the case of cancer and lardaceous disease of the capsules, it may be just possible that portions remain healthy, and are sufficient to fulfil the usual healthy functions of the organs; and this suggestion is borne out by analogy with the liver, kidneys, etc.

Symptoms.—Those of anæmia. The patient complains of weakness and debility, and progressive impairment in the general health. The skin, especially about the face and neck, assumes a dingy brownish appearance. The pulse is feeble, and the patient complains of persistent and unconquerable listlessness and disinclination for any exertion. The appetite is capricious, and vomiting is not unfrequent. It must be again remarked that the peculiar coloration of the skin, which Addison considered to be due to disease of the supra-renal capsules, often occurs without any disease in these organs being detected. It is no uncommon thing for the skin of pregnant women to become of a dusky hue from deposit of pigment in the rete mucosum. This may be due to the temporary pressure on the solar plexus, or on other portions of the sympathetic system of nerves in the abdomen, by the gravid uterus, and which may temporarily produce the same effect that the chronic disease of the organs permanently maintains. Be this as it may, the colour during pregnancy in most cases disappears with delivery.

Treatment.—This will chiefly be directed to relieve the anæmic condition of the patient, and to improve the general health by proper hygienic measures and diet. Most cases end fatally.

DISEASES OF THE SKIN.

The diseases now to be briefly considered affect the cutaneous surface of the body. They require for their cure both local and constitutional treatment. In many cases they have a tendency to become chronic, and are then very troublesome to cure. The constitutional treatment will consist for the most part in allowing a nourishing diet, and in the administration of the preparations of iron, arsenic, mercury, iodine, etc. The local treatment will require the application of simple lotions to allay the intolerable itching which so often attends the course of these affections, or the use of ointments which contain certain active ingredients, and which, as in the case of the order Parasitici, destroy the insect or growth which is the cause of the disease. Nearly every dermatologist has a particular classification of his own, but for the sake of convenience, these diseases will be considered in the following order:—

PAPULÆ.

Examples—LICHEN; PRURIGO.

General Characters.—Simple elevations of the cuticle, sometimes inflamed.

LICHEN.—This disease is characterised by the presence of inflamed elevations of the cuticle, accompanied with intense itching, and sometimes occurring on the back of the hands, at other times scattered over the shoulders and other parts of the body. Lichen may result from cold, over-feeding, debility, etc.

Lichen strophulus, or Red Gum, the earliest eruption of infants. Lichen sometimes occurs on the hands of grocers, bakers, and washerwomen. Grocers' itch is *Lichen agrius*. By some, bakers' and grocers' itch are considered to be forms of eczema.

Treatment.—The treatment will consist in the use of mild laxatives, restricted diet, and warm baths. Bismuth and colchicum may be sometimes given with advantage to those of a gouty diathesis. The irritation may be allayed by the use of the common lead lotion, to which a few drops of hydrocyanic acid have been added.

PRURIGO.—The aged are very frequently troubled with this affection, and as it is attended with intense itching, the annoyance is very great. The eruption consists in slightly-raised papules of the same colour as the skin, distinct, and covered at their tops with small black points, due to the scratching to relieve the intense itching.

There are several varieties—*Prurigo mitis, Prurigo formicans, Prurigo senilis.* The nates and the outside of the thighs are the parts usually affected in old people. Care must be taken not to confound this disease with those of a parasitic origin. It is often most intractable, no method of treatment seeming to have much power over it. The application of dilute acetic acid, or the use of the lotion recommended in the last case, will often relieve the intense itching. The diet must be unstimulating. Sponging the parts affected with apple vinegar, allowing it to dry, and then smearing them with citrine ointment, has been recommended by Dr Bowling of Kentucky. A carefully regulated diet, gentle laxatives, the internal use of liquor potassæ, arsenic, iron, cod-liver oil, etc., are the means generally employed.

Prurigo must not be confounded with lichen, scabies, and strophulus. Strophulus occurs most frequently in the young; prurigo in the aged. The papules of the former are redder or whiter than the surrounding skin, and the black crusts are wanting. In lichen the papules are small, aggregated together, acuminated, of a red colour, and covered by grey or furfuraceous crusts. In scabies the presence of the acarian furrows may be detected by a lens. The eruption of scabies may be detected between the fingers, that of prurigo *never (Squire).*

VESICULÆ.

Examples.—HERPES; SUDAMINA; ECZEMA.

General Characters.—Small elevations of the cuticle on an inflamed base, containing serum.

HERPES.—Clusters of vesicles on a common inflamed base, the clusters being frequently separated by healthy skin. After a time the vesicles dry up and form scabs. There are several varieties.

Herpes Labialis.—Appearing on the lips and nostrils, due to catarrh and indigestion, and sometimes occurring in the course of ephemeral fever, typhoid and relapsing fever.

Herpes Preputialis, occurring on the glans penis, has been treated for syphilis.

Herpes Circinatus may occur on the scalp, and be treated for ring-worm.

Herpes Zoster, or the shingles, is usually confined to one side of the body, generally the right. It is accompanied with a deep-seated internal pain in the course of one of the intercostal nerves. The pain, probably due to some nervous irritation, is sometimes present for a day or two before the appearance of the eruption. Dr Liveing gives the following points of distinction between *Herpes Labialis* and *Herpes Zoster*:—

1. In *Herpes Zoster* we have often several clusters of vesicles in succession; in *Herpes Labialis*, seldom more than one crop.
2. *Herpes Zoster* commonly appears only once in the life of an individual; *Herpes Labialis* may recur any number of times.
3. *Herpes Zoster* is associated with a morbid condition of some cerebro-spinal nerve; *Herpes Labialis* generally occurs with some catarrhal affection, pneumonia, etc.
4. Neuralgic pains precede the eruption of *Herpes Zoster*, and may remain long after its disappearance; this is not the case with *Herpes Labialis*.
5. *Herpes Zoster* is often unilateral, but *Herpes Labialis* is not so.

Mild purgation and regulation of the diet is all that will be required by way of treatment. The neuralgia in *Herpes Zoster* may be treated by the subcutaneous injection of morphia. In *Herpes Labialis* the scabs may be removed by a bread-poultice and a little zinc ointment smeared over the part.

SUDAMINA.—Minute vesicles occurring during the course of some acute diseases which are attended with profuse sweating. The eruption is identical with that appearing in the disease described by some writers as *Miliaria*, and considered by them as a special fever.

ECZEMA.—Varieties—*Eczema Simplex, Eczema Rubrum, Eczema Impetiginodes, Eczema Chronicum.*

The vesicles in eczema are very minute and very numerous, crowded together in broad irregular patches, attended with much tingling, heat, and itching, and a copious discharge. Desquamation then takes place, the scabs coming off in thin flaky crusts. Eczema is most frequently seen on the backs of the hands, the skin over the shin, the nape of the neck, and at the back of the ear. It may, however, extend over the whole body, with a strong tendency to become chronic. Eczema rubrum of the face may be mistaken for erysipelas. It is often constitutional, and is sometimes hereditary.

Treatment.—The local treatment will consist in the use of soothing applications, among which may be mentioned cod-liver oil.

If the disease have not become chronic, besides the local treatment, saline aperients will be required. Should the disease, however, assume a chronic form, it will be necessary to try the effects of arsenic. Strict attention must also be paid to the regulation of the diet. In gouty dyspeptic patients treat the diathesis; all specifics are useless without common sense. Many a case may be cured by simple attention to the diet where a patient's stomach had previously been made the receptacle for almost every known drug. The late Professor Bennett recommended the application of weak alkaline lotions, constantly applied.

The following Table may assist in the Diagnosis of Eczema.

LICHEN.	PSORIASIS.	ERYTHEMA INTERTRIGO.	HERPES.	IMPETIGO.	FOLIACEOUS PEMPHIGUS.	ECZEMA.
Secretion small. Crusts minute, dry, and greyish. Skin harsh, dry, and thickened. In eczema, the skin is smooth, thinned, and moist.	Crusts dry, white, laminated, and nacreous (pearl-like). Skin inflamed, dry, elevated, creased, tawny-red. Situation of eruption, the loins, knees, and elbows.	Secretion thin; generally situated in some fold of the skin. Readily yields to treatment.	Vesicles larger than in eczema. Occurs in circumscribed patches. Areolæ inflamed, the crusts adherent, not exfoliations as in eczema.	The crusts thicker than in eczema, nodulated when removed; base is found in a state of suppuration. The disease in its early stage is pustular.	Exfoliations large. Secretion thin. Bullæ may also be present.	Transparent vesicles varying in size, sometimes as large as a split pea. Sometimes a multitude of red cracks in the skin. Moist slightly excoriated reddened surface. Secretion abundant, clear, plastic serum, concreting into buff-coloured, flaky crusts, ending in scaly desquamation. Occurs at the bends of joints, but may also extend to other parts of the body.

Note.—Eczema of the scalp so closely resembles pityriasis that the diagnosis is made with difficulty. The scales of pityriasis are dryer and more opaque than in eczema. Guard against mistaking scabies for eczema of the hand: the former generally occurs between the fingers; the latter along the fingers, both back and front.

PARASITICI.

Examples. — 1. TINEA TONURANS, TINEA FAVOSA, TINEA DECALVENS, TINEA SYCOSIS, PLICA POLONICA AND CHLOASMA.—2. SCABIES.

General Characters.—They are all contagious. Those placed under the first section are due to the presence of vegetable growths; that under the second, to an insect. The presence of the gooseberry bug under the skin has been mistaken for eczema.

TINEA TONSURANS.—The parasite in this case is the *Trichophyton Tonsurans.* The disease has received several names—*Porrigo Scutulata, Herpes Tonsurans, Herpes Circinatus, Trichosis Furfuracea,* and lastly, *Ring-worm.*

Circular red patches, with numerous minute pustules, crowded at the circumference of the patch, characterise the disease. The hair breaks off about a line from the scalp, giving the idea that the part has been shaved—hence the name.

Ring-worm may occur on other parts of the body besides the scalp.

TINEA FAVOSA.—*Porrigo Favosa, Tavus, Tinea Lupinosa,* characterised by the presence of bright or sulphur-yellow unimbilicated scabs, resembling a honey-comb. The disease is accompanied with the peculiar odour of mice. The parasite is the *Achorion Schönleinii.* It most frequently attacks the scalp.

TINEA DECALVENS.—The presence of bald patches on the scalp, more or less circular, due to the destruction of the hair by a parasitic fungus, the *Microsporon Audouini.* This affection is also known by the following synonyms —*Porrigo Decalvens* or *Alopecia Circumscripta.*

TINEA SYCOSIS.—This occurs on the chin of men, and is connected with a vegetable parasite, the *Microsporon Metagrophytes,* or the *Tinea Sycosis,* in the hair follicles of the beard.

Treatment.—The treatment to be adopted is the same. Perfect cleanliness; removal of the hairs by epilation or shaving will be required. The scabs must be removed by the application of soft poultices; and then a strong solution of sulphurous acid, or ointments containing substances to destroy the vegetable growths, must be applied. For this purpose the ammonia chloride of mercury ointment, or the unguentum picis liquidum, will be found useful.

Plica Polonica is a disease not unlike ring-worm, seldom seen in England, but common enough in Poland. The parasite, *Trichophyton Sporuloides.*

Chloasma.—Pityriasis versicolor, or liver spot. It generally occurs on the chest or shoulders, in patches varying in size, and of a bright liver or reddish-brown colour. It is said to be due to a parasitic fungus, the *Microsporon Furfur.* The application of the sulphurous acid lotion will in most cases effect a cure.

Table showing Diagnostic Differences in the following Diseases :—

PITYRIASIS.	PSORIASIS CIRCINATA.	LICHEN CIRCUMSCRIPTUS.	HERPES CIRCINATUS.
Patches with irregular rounded smooth margins; tawny redness all over the patch.	Scales larger and thicker, with a pearly lustre, the ring raised; the redness has a tawny hue, the centre either healthy or of a tawny yellow.	Patches irregularly circular, the scales thicker and more harsh. Centre and the margins equally rugous.	Small, rounded, rosy-red patches slightly raised above the surrounding skin. The surface becomes rough and scurfy, and the hairs on the patch are easily broken.

2. Scabies.—Scabies, psora, or the itch, depends on the irritation of an animalcule, or kind of mite, the *Scabiei*, or the *Sarcoptes Scabiei*, which burrows in the cuticle. It gives rise to the presence of vesicles, which appear most frequently on the hands, especially between the fingers. The vesicles are surrounded by a rosy-coloured base. Sometimes by the aid of a lense the track of the animal under the skin may be seen. The itching is intense, warmth in bed generally increasing it.

Treatment.—The treatment will consist in the inunction of the common sulphur ointment. This is best done before a fire.

Table giving the Animal and Vegetable Parasites and the Diseases which they Produce:—

ANIMAL PARASITES.	VEGETABLE PARASITES.
1. Lice (Phthirriasis—the lousy disease).—Varieties of lice are *Pediculus Capitis; Pediculus Corporis; Pediculus Pubis, and Phthirius Inguinalis* or *Crab Louse.*	1. Achorion Schönleinii—Favus (Syn. *Porrigo Favosa; Tinea Favosa*).
2. Fleas, Bugs, etc.	2. Tricophyton Tonsurans—(*a*) Ring-worm (Syn. *Tinea Tonsurans; Tinea Circinata; Herpes Circinatus*). (*b*) Tinea Sycosis (Syn. *Parasitic Mentagra; Barbers' Itch*, etc.
3. Mites—(*a*) Acarus Scabiei, the itch insect; (*b*) Septus (or Acarus) Autumnalis, the Harvest Bug; (*c*) Demodex Folliculorum.	3. Microsporon Audouini—Alopecia Areata, the parasitic form.
4. Dranunculus (Syn. *Filaria Medinensis*), Guinea Worm.	4. Microsporon Furfur—Pityriasis Versicolor.
	5. Chionyphe Carteri—The Madura foot (Syn. *Myatoma*).

—Dr Wyllie's *Lecture Notes.*

BULLÆA.

Examples.—PEMPHIGUS; POMPHOLYX.

General Characters.—Vesicles of large size.

PEMPHIGUS is attended with more or less fever, and by the presence of bullæ or blebs on the surface of the body. When the vesicles burst, they are succeeded by thin brownish-coloured crusts. This disease may last from one to four weeks, or even longer. A form of pemphigus not unfrequently occurs in babies, and is held by some to be of a purely syphilitic origin. In them it may end fatally.

Pemphigus must not be confounded with any of the following:—

HERPES ZOSTER.	RUPIA.	ECTHYMA.	IMPETIGO.	ERYSIPELAS.
Tendency to encircle the body; the blebs small, seldom larger than a pea.	Blebs few in number; the crusts thick, conical, resting on ulcers.	Crusts black and thick, and contain purulent fluid.	Crusts thick, yellow, and brittle.	Blebs of irregular outline; the surrounding skin inflamed and brawny.

POMPHOLYX, in which the vesicles are as large as nuts or small walnuts, unattended with inflammatory symptoms.

The **Treatment** of the above will consist in the use of alterative medicines, and the allowance of a nutritious diet.

PUSTULÆ.

Examples.—ECTHYMA; IMPETIGO; ACNE; RUPIA.

General Characters. — Inflamed elevations of the cuticle containing pus, followed by scabbing, and leaving cicatrices.

ECTHYMA usually occurs in syphilitic and cachectic subjects. The pustules are large and distinct, situate on inflamed and hard bases. After a time they burst, and are succeeded by thick dark crusts or scabs.

IMPETIGO is a non-contagious pustular eruption occurring on different parts of the body. The pustules are generally in clusters, and form thick prominent rough crusts. The disease has been divided into two varieties —*Impetigo Figurato* and *Impetigo Sparsa*.

In the **Treatment** of the above, mild saline laxatives will be necessary, together with the application of the oxide of zinc ointment to the pustules after the removal of the scabs by means of a poultice. Tonics may sometimes be required in the latter stages of the affection.

ACNE.—Acne occurs in most cases on the shoulders, or on the face of young persons about the age of puberty, and consists in inflammation and suppuration of the subaceous follicles. This condition is probably due to some morbid state of the secretions induced by errors in diet, and a want of attention to the regular action of the bowels. Three varieties of this disease have been described by Willan—*Acne Indurata*, *Acne Rosacea*, and *Acne Simplex*.

Aperients, tonics, fresh air, and cleanliness will meet the requirements of the case.

RUPIA.—Rupia, sometimes classed with the bullæ, is in most cases an evidence of a syphilitic or cachectic taint. It is remarkable as regards the formation of the scabs, which are conical, and have been compared to limpet shells. A deep ulcer is most frequently found under each crust.

The **Treatment** of rupia will in part be directed to the removal, if possible, of the cachectic condition of the patient, and in part to the healing of the subsequent ulceration after the fall of the scab. Tonics, the iodide of potassium, or iodide of iron, may be used, together with careful attention to hygienic rules, and the allowance of a nutritious diet. A little zinc ointment to the ulceration will be all that is required by way of local treatment.

SQUAMÆ.

Examples.—LEPRA; PSORIASIS; PITYRIASIS; ICTHYOSIS.

General Characters.—The formation of scales of morbid cuticle, generally in patches of a whitish colour, and covering dry red surfaces.

LEPRA.—This disease, which is non-contagious, usually occurs on the limbs at the bend of the joints in annular patches, spreading at their circumferences, and healing towards the centre. It often has a syphilitic origin.

The **Treatment** will demand the use of alkalies, arsenic, and in the syphilitic variety, known by its copper-coloured appearance, mercury will most probably be necessary to effect its removal.

PSORIASIS is more diffused than lepra, and is chiefly found on the palms of the hands and soles of the feet. It is also frequently of syphilitic origin. Psoriasis is also known as "dry tetter," in contradistinction to herpes, or "wet tetter." It is non-contagious and requires the same sort of treatment as lepra, with which it is frequently confounded.

PITYRIASIS, in which the scales are very small. It chiefly occurs on the scalp in the form of dandriff or scurf. The best treatment for this is the use of the mild citrine ointment, made up with a little pleasant pomade

Icthyosis, or fish skin, is a disease marked by the presence of fish-like scales. It is congenital and incurable. There is not unfrequently some deposit of pigment under the cuticle.

EXANTHEMATA.

Examples.—Erythema; Roseola; Urticaria.

General Characters.—Irregular superficial red patches, disappearing on pressure, and terminating in desquamation.

Erythema.—The most curious kind is that known as *Erythema nodosum*, appearing as oval red swellings along the shins and ulnæ of girls and young women, continuing about a fortnight, and then going off, leaving the appearance of a bruise.

The use of iron and bark will be required in the treatment of the above.

Roseola.—*Roseola æstiva* occurring in children during the summer-time, with slight febricula. It is non-contagious. The patches are larger than those of measles, but smaller than those of scarlet fever, for which diseases it may be frequently mistaken. Mild laxatives will be all that is necessary.

Urticaria, or nettle-rash, is a non-contagious eruption of the skin, characterised by the presence of prominent patches, or wheals. It usually arises from irritation of the stomach and bowels by shell-fish, or some other indigestible food. Nettle-rash may be accompanied with vomiting and purging. The itching, which more or less attends this disease, is sometimes almost unbearable. The duration of urticaria is short.

Warm baths and simple saline medicines are alone required. If the cause be due to stomach derangement, an emetic or purgative may be necessary.

TUBERCULÆ.

Examples. — ELEPHANTIASIS; MOLLUSCUM; LUPUS; FRAMBŒSIA; KELOID.

General Characters.—Hard indolent elevations of the skin, sometimes ulcerating at their summits, sometimes partially suppurating.

ELEPHANTIASIS, divided into *Elephantiasis græcorum* and *Elephantiasis arabicum.* The former, or true leprosy, is a non-contagious, hereditary, and fatal disease, consisting in tubercular purplish-coloured patches, which tend to ulcerate. The latter, Elephantiasis arabicum, or Barbadoes leg, is characterised by swelling and induration of the skin and sub-cutaneous areolar tissue. The lower extremities are the parts most frequently attacked. Both these diseases are peculiar to hot countries.

MOLLUSCUM CONTAGIOSUM. — This disease is fortunately very rare, and must not be confounded with Molluscum fibrosum. Molluscum contagiosum is characterised by the presence of rounded elevations on the skin, varying in size from that of a mustard seed to that of a walnut, or even larger. The elevations consist of little cysts filled with a hard or soft sebaceous matter. The disease is unattended with constitutional disturbance.

The **Treatment** will consist in squeezing out the contents of the cysts, and then applying a point of nitrate of silver. The administration of tonics will also be necessary.

LUPUS, frequently occurring on the face, especially the nose, and ulcerating deeply, as well as superficially destroying the skin and cartilages. It may be confounded with cancer and syphilis. Lupus not unfrequently occurs in young scrofulous persons.

Treatment.—Tonics, cod-liver oil, and the application of caustics externally. The best caustic for this purpose is the chloride of zinc. The ulcers may be removed with a scraper.

FRAMBŒSIA, or yaws, is a tubercular disease common in Africa, the West Indies, and America. It may attack any part of the body, and is most difficult to remove.

KELOID—*Chelonia*, *Cheloidea*, or *Cancroide*,—is a disease of a fibrous nature, in which the skin acquires the appearance of the shell of the tortoise.

MEDICINAL RASHES.—These may be produced by the bromides, by chloral hydrate, arsenic, copaiba, belladonna, mercury, and several other drugs.

The **Treatment** consists in discontinuing the admini stration of the drug for a time.

DIETS FOR VARIOUS DISEASES.

DIET FOR DIABETES (PAVY).

May eat and drink—	*May not eat or drink—*
Butchers' meat of all kinds. Bacon, ham, or other cured meats. Poultry, game. Shell-fish, and fish of all kinds. Animal soups not thickened. Almond, bran or gluten bread,* cheese, cream cheese, butter, cream. Greens, spinach, *turnip tops*, *French beans*, *Brussels sprouts*, *cauliflower*, *broccoli*, *cabbage*, *asparagus*, *sea kale*, *vegetable marrow*, mushrooms, water-cress, mustard and cress, cucumber, lettuce, endive, radishes, celery. Vinegar, oil, pickles. Jelly, not sweetened, savoury jelly, custard. Nuts, except chestnuts, olives. Tea, coffee, cocoa from nibs, dry sherry, claret, dry sauterne, Burgundy, chablis, hock, brandy, soda water, *Burton ale.*	Sugar in any form, wheaten bread and biscuits, rice, arrow-root, sago, tapioca, macaroni, vermicelli, potatoes, carrots, parsnips, beetroot, peas, Spanish onions, pastry or puddings of all kinds, fruits of all kinds, milk, sweet ales mild or old, porter or stout, cider, all sweet wines, sparkling wines, and port wines, unless sparingly, liqueurs.

DIET FOR FEVER AND INFLAMMATORY DISEASES.—Beef-tea, mutton, veal or chicken broth, whey, jelly, arrow-root, and custards, barley, rice or gum water, and drinks made of the juice of lemons or oranges. Then fish may be given during convalescence, especially whiting; gradually chicken, game, and butchers' meat may be allowed, mutton being first given.

DIET FOR GASTRIC ULCER. — Boiled milk, milk and lime water, or soda water, milk and isinglass or ground rice.

DIET FOR GOUT.—Chiefly vegetable, and the light dry wines, if they agree, avoiding port, stout, and strong sweet ale.

---

* These may be obtained of Messrs Blatchley, 167 Oxford Street, London.

The articles of diet in italics are to be used sparingly.

PRESCRIPTIONS.

ANTHELMINTICS AND CATHARTICS.

1. ℞ Magnesiæ Sulphatis, ʒii ; Mannæ, ʒiiss ; Tincturæ Sennæ, ʒii ; Infusam Sennæ ad ℥iss. Fiat mistura.—*Common Black Draught.*

2. ℞ Magnesiæ Sulphatis, ℥iss ; Magnesiæ Carbonatis, ʒii ; Liquoris Ammoniæ Acetatis, ℥i ; Aquam Menthæ Piperitæ ad ʒviii. Fiat mistura. Sumat ℥i ter in die.

3. ℞ Magnesiæ Sulphatis, ʒii ; Ferri Sulphatis, gr. iv ; Acidi Sulphurici Diluti, ♏xv ; Extracti Quassiæ, gr. xx ; Aquæ Pimentæ, ℥iss. Fiat haustus.—*In Constipation with Debility.*

4. ℞ Extracti Aloës Barbadensis, gr. iii ; Extracti Nucis Vomicæ, gr. ss ; Mastiches Gummi, gr. ss. Misce. Fiat pilula. Sumat unam omni nocte.—*In Confirmed Constipation in Women.*

5. ℞ Extracti Filicis Liquidi, ʒss ; Mellis, ʒiii. Misce.—*In Tape Worm.*

6. ℞ Santonini, gr. ii–iii ; Pulveris Scammoniæ, gr. iii–v. Misce.—*In Round Worm.*

ANTISPASMODICS.

7. ℞ Spiritûs Ætheris, ℥iss ; Tincturæ Opii, ʒi ; Tincturæ Castorei, ʒii ; Aquam Menthæ Piperitæ ad ℥vi. Misce. One ounce every four hours.—*In Flatulence.*

8. ℞ Tincturæ Assafœtidæ, ʒii ; Spiritûs Ammoniæ Aromatici, ʒiii ; Tincturæ Chiratæ, ʒiii ; Aquam Menthæ Piperitæ ad ʒvi. Misce. One ounce every four hours.

9. ℞ Tincturæ Valerianæ, ʒiii; Olei Cajuputi, ♏xii; Spiritûs Chloroformi, ʒiss; Infusum Gentianæ Compositum ad ℥vi. Misce. Sumat ℥i ter in die.—*In Hysteria.*

10. ℞ Acidi Hydrochlorici Diluti, ʒiss; Olei Cajuputi, ♏xii; Infusum Gentianæ Compositum ad ℥vi. Misce. Sumat ℥i ter in die.—*A good Mixture in Acid Dyspepsia given before Meals.*

ASTRINGENTS.

11. ℞ Pilulæ Plumbi cum Opio, gr. iv. To be taken every two or three hours.

12. ℞ Magnesiæ Sulphatis, ℥ss; Tincturæ Digitalis, ʒi; Acidi Gallici, ʒi; Extracti Ergotæ Liquidi, ʒii; Infusum Rosæ Acidum ad ℥viii. Misce. Sumat ℥i tertiâquâque horâ vel quârtaquâque horâ.—*Dobell's Mixture for Hæmoptysis.*

13. ℞ Tincturæ Opii, ʒi; Olei Cajuputi, ♏xii; Tincturæ Rhei, ℥ss; Misturam Cretæ Aromaticam ad ℥vi. Misce. ℥i post sedes liquidas.—*In simple painful Diarrhœa.*

14. ℞ Tincturæ Opii, ʒi; Acidi Sulphurici Diluti, ʒii; Tincturæ Cinamomi, ʒiii; Aquam Camphoram ad ℥vi. Sumat ℥i bis vel tertiâquâque horâ. *In severe Diarrhœa and early stages of Cholera.*

DIAPHORETICS AND DIURETICS.

15. ℞ Potassæ Nitratis, gr. 120; Acidi Nitrici Diluti, ʒi; Tincturæ Digitalis, ʒss; Aquam Rosam ad ℥vi. Misce. One ounce every four hours.—*In Relapsing Fever.* (Murchison.)

16. ℞ Potassæ Nitratis, gr. 60; Vini Ipecacuanhæ, ʒiss; Liquoris Ammoniæ Acetatis, ℥ss; Misturam Amygdalam ad ℥vi. Misce. A table-spoonful every four hours.—*In Catarrh and early stages of Fever.*

17. ℞ Tincturæ Guaiaci Ammoniatæ, ʒiii–vi; Mucilaginis Tragacanthæ, ℥iii. Mix, and then add Infusi Senagæ, ℥viii. Three table-spoonfuls to be taken every four hours.—*In Chronic Bronchitis. Is diaphoretic, diuretic, stimulant, and expectorant.*

18. ℞ Ammoniæ Sesquicarbonatis, gr. v; Spiritûs Ætheris Chlorici, ♏x; Vini Colchici, ♏v; Liquoris Ammoniæ Acetatis, ʒiii; Misturam Acaciæ ad ℥iss. Misce et fiat haustus quartâquâque horâ sumendus.—*Useful in Pneumonia complicated with Gout.*

19. ℞ Potassæ Acetatis, ʒss; Aceti Scillæ, ʒss; Spiritûs Ætheris Nitrosi, ♏xx; Tincturæ Digitalis, ♏v; Decocti Scoparii, ℥iss. Misce. Ter die sumendus.—*In Dropsy due to Heart Disease.*

EMETICS AND EXPECTORANTS.

20. ℞ Cupri Sulphatis, gr. x; Aquæ, ℥iii. Misce. Fiat haustus emeticus, statim sumendus.—*A quick Emetic. May be repeated.*

21. ℞ Zinci Sulphatis, ʒss; Aquæ, ℥iii. Misce.—*Ut supra.*

22. ℞ Vini Ipecacuanhæ, ʒi. Statim sumendus.—*For a Child.*

23. ℞ Tincturæ Scillæ, ♏iv; Tincturæ Camphoræ Compositæ, ʒss; Syrupi Simplicis, ʒss; Misturæ Amygdalæ, ʒvii. Misce. Ter die sumendus.

24. ℞ Ammoniæ Sesquicarbonatis, gr. xi; Tincturæ Scillæ, ʒiss; Decocti Senegæ, ℥viii. Misce. Sumat ℥i omnibus sextis horis.—*In Chronic Bronchitis.*

25. ℞ Tincturæ Scillæ, ʒi; Acidi Phosphorici Diluti, ʒss; Extracti Conii, gr. x; Syrupi Papaveris, ℥ss; Infusi Cinchonæ, ℥iv. Misce. Sumat ℥i bis terve die.—*In Chronic Catarrh.*

26. ℞ Vini Ipecacuanhæ, ʒii; Liquoris Ammoniæ Acetatis, ℥ss; Syrupi Simplicis, ℥ss; Misturam Amygdalam ad ℥vi. Misce. Sumat ℥i ter in die.—*In Acute Bronchitis.*

27. ℞ Succi Conii, ʒi; Acidi Hydrocyanici Diluti, ♏x; Vini Ipecacuanhæ, ʒi; Syrupi, ℥ss; Aquam ad ℥iss. Misce. Sumat ʒi quartâquâque horâ.—*In Whooping-Cough, for a Child two years old.*

GARGLES AND INHALATIONS.

28. ℞ Aluminis Exsicati, ʒii; Infusum Rosæ Acidum ad ℥viii. Misce. Fiat gargarisma.—*In Relaxed Throat.*

29. ℞ Liquoris Hydrargyri Bichloridi, ℥viii; Extracti Conii, ʒi. Misce. Fiat gargarisma.—*In Syphilitic Sore Throat.*

30. ℞ Boracis, ʒii; Glycerini, ℥i. Misce. To be painted over the mouth.—*Useful in Aphthæ.*

31. ℞ Creasoti, ♏xxx; Aquæ Bullientis, ℥viii. *Inhale in Phthisis, etc.*

32. ℞ Liquoris Sodæ Chloratæ, ʒvi; Aquam ad ℥viii. Misce. Fiat gargarisma. *In Scarlet Fever, Profuse Salivation, etc.*

LINIMENTS, LOTIONS, ETC.

33. ℞ Olei Succini, ℥ss; Olei Olivæ, ℥ss; Olei Caryophilli, ʒi. Misce. Fiat linimentum.—*A substitute for Roche's Embrocation.*

34. ℞ Aluminis Exsicati, gr. iv; Aquæ Rosæ, ℥i. Misce.—*To be dropped into the Eye as a mild astringent.*

35. ℞ Linimenti Belladonnæ, Linimenti Camphoræ Compositi, ā ā ℥i. Misce.—*In Lumbago, Rheumatism, etc.*

36. ℞ Linimenti Hydrargyri, ℥ii; Linimenti Belladonnæ, Linimenti Opii, ā ā ℥i. Misce.—*In Syphilitic Nodes, etc.*

37. ℞ Spiritûs Rectificati, ℥i; Acidi Acetici Diluti, ℥i; Ammoniæ Hydrochloratis, ʒii; Aquam ad ℥viii. Misce. Fiat lotio.—*Useful in Orchitis, etc.*

NARCOTICS AND SEDATIVES.

38. ℞ Acidi Phosphorici Diluti, ʒi; Liquoris Morphinæ Hydrochloratis, ʒiss; Acidi Hydrocyanici Diluti, ♏xii; Syrupi Rhœados, ℥i; Aquam ad ℥iss. Misce. Sumat ʒi tusse urgenti. —*A valuable Cough Mixture.*

39. ℞ Extracti Opii, gr. i; Extracti Belladonnæ, gr. $\frac{1}{3}$; Butyri Cacao, gr. xx. Mix into a Suppository.—*In Irritation of Bladder or Rectum.*

40. ℞ Extracti Opii, gr. i; Pulveris Capsici, gr. i; Extracti Gentianæ, gr. iii. Misce. Divide into two pills.—*When Opium alone is not retained by the Stomach.*

41. ℞ Tincturæ Digitalis, ʒi; Tincturæ Cardamomi Compositæ, ʒvi; Acidi Hydrocyanici Diluti, ♏xx; Aquam Chloroformum ad ℥vi. Misce. ℥i quartâquâque horâ sumendus.

REFRIGERANTS AND SALINES.

42. ℞ Quininæ Sulphatis, gr. xii; Pulveris Digitalis, gr. xii; Pulveris Opii, gr. vi. Misce. Divide into twelve powders; one to be taken every four hours.—*Niemeyer's Powders in Acute Phthisis with high temperature.*

43. ℞ Liquoris Ammoniæ Acetatis, ʒvi; Spiritûs Ammoniæ Aromatici, ʒiii; Syrupi Limonis, ℥i; Aquam ad ℥vi. Misce. Sumat ℥i tertiâquâque horâ.—*In slight Fever.*

44. ℞ Potassæ Tartratis Acidi, ℥i; Olei Limonis, ♏xv; Sacchari Albi, ℥ii; Aquæ Bullientis, Oii. Misce. When cold take a wine glassful occasionally.

45. ℞ Potassæ Chloratis, ʒi; Syrupi Limonis, ℥iii; Aquæ ad Oi.—*As a drink in Scarlet Fever.*

STIMULANTS.

46. ℞ Spiritûs Ammoniæ Aromatici, ʒi; Tincturæ Lavandulæ Compositæ, ʒii; Syrupi Zingiberis, ℥ss; Aquam ad ℥i. Sumat ℥i ter in die.

47. ℞ Misturæ Spiritûs Vini Gallici, B.P.; Sumat ℥i quartâquâque horâ.—*In Debility.*

48. ℞ Liquoris Ammoniæ Acetatis, ℥ss; Spiritûs Ammoniæ Aromatici, ʒiii; Tincturæ Cardamomi Compositæ, ʒiii; Syrupi Zingiberis, ℥ss; Infusum Gentianæ Compositum ad ℥vi. Misce. Sumat ℥i pro re nata.—"A pick me up."

49. ℞ Ammoniæ Carbonatis, ʒss; Tincturæ Aurantii, ℥ss; Infusum Cinchonæ ad ℥vi. Misce. Two table-spoonfuls every four hours.

TONICS.

50. ℞ Tincturæ Ferri Perchloridi, ʒss; Potassæ Chloratis, ʒi; Syrupi, ℥ss; Aquam ad ℥vi. Misce. Two table-spoonfuls every four hours.—*In Diphtheria.*

51. ℞ Misturæ Ferri Compositæ, ℥vi. Sumat ℥i ter in die.—*In cases requiring a non-astringent preparation of Iron.*

52. ℞ Tincturæ Ferri Perchloridi, ʒi; Glycerini, ℥ss; Aquam ad ℥vi. Misce. Sumat ℥i ter die.—*The Glycerine disguises the Iron.*

53. ℞ Quininæ Sulphatis, gr. xii; Infusi Rosæ Acidi, ℥vi. Misce. Sumat ℥i ter in die.

INDEX.

PAGE
Abscess of the Liver 317
Acne 491
Acute Ascending Paralysis 436
  " Bright's Disease 441
  " Bronchitis 202
  " Desquamative Nephritis 442
  " Encephalitis 344
  " Farcy 141
  " Gastritis 282
  " Glanders 141
  " Peritonitis 309
  " Rheumatism 145
  " Yellow Atrophy of the Liver 328
Ague 116
Albuminuria 441
Albumen, Tests for 468
Algide Cholera 93
  " Yellow Fever 63
Amyloid Degeneration of Liver 325
Anæmia 164
  " Progressive Pernicious 164
Anasarca 10
Anatomical Relations of Heart, 253
Aneurism, Thoracic 277
Angina Pectoris 275
Anginosa Scarlatina 81

PAGE
Anthelmintics 497
Anthrax 142
Antispasmodics 497
Aorta, Aneurism of 277
Aphasia 368
Aphemia 369
Aphthæ of the Mouth 280
Apoplexy 360
Ascarides 305
Ascites 312
Asiatic Cholera 93
Asthma 210
  " Renal 459
Astringents 498
Ataxy 392
  " Walk 419
Atrophy 4
  " of the Heart 268
  " of the Liver 328
Auditory Vertigo 401

Bakers' Itch 482
Bell's Palsy 426
Biliary Calculi 338
  " Ducts, Inflammation of 337
Bile, Tests for 468
Bilious Remittent Fever 121
Bites of Rabid Animals 136
Bleeding from the Stomach 9
Blood, Tests for 469
Blue Disease 272

PAGE
Bothriocephalus Latus . 306
Bowels, Obstruction of . 303
" Relaxation of . 299
Brain, Abscess of . . 349
" Dropsy of . . 350
" Hæmorrhage into the . . . 360
" Inflammation of the 344
" Ramollissement of 349
Bright's Disease . . 441
Bronchiectasis . . 208
Bronchitis . . . 202
" Acute . . . 202
" Chronic . . 207
" Plastic . . . 209
Broncho-Pneumonia . 225
Bullæ 490

Calculi, Biliary . . 338
" Urinary . . 477
Cancer of the Kidney . 474
" Liver . . . 326
" Lungs . . . 241
" Œsophagus . . 282
" Stomach . . 293
Cancrum Oris . . 280
Carcinoma . . . 176
Cardiac Valvular Disease 254
Catalepsy . . . 411
Catarrh 181
Catarrhal Pneumonia . 225
Cathartics . . . 497
Cerebellar Tumours . 368
Cerebral Hæmorrhage . 360
" Paralysis . . 417
Cerebritis . . . 344
Cerebro-Spinal Meningitis 61
Chalk Stones . . 153
Charbon . . . 142
Charcot's Joint Disease . 394
Cheloidea . . . 495
Cheyne Stoke's Respiration 260
Chicken-Pox . . . 80
Chloasma . . . 488
Chlorides, Tests for . 468
Chlorosis . . . 164

PAGE
Cholera, Malignant . 93
" Summer . . 98
Chorea 387
Chronic Bright's Disease 449
" Bronchitis . . 207
" Farcy . . . 141
" Gastritis . . 282
" Glanders . . 141
" Gout . . . 157
" Peritonitis . . 311
" Pneumonia . . 226
" Rheumatism . . 147
Chyluria . . . 472
Cirrhosis of the Liver . 320
" Lung . . . 311
Clergyman's Sore Throat 185
Cæcitis 298
Cæcum, Inflammation of 298
Collapse of Lung . . 204
Colic 300
Colica Pictonum . . 301
Colloid Cancer . . 176
Concentric Cardiac Hypertrophy . . 266
Congestion of the Lungs 212
Constipation . . . 302
Convulsions, Uræmic . 463
Corrigan's Pulse . . 259
Coryza 180
Coup de Soleil . . 370
Cow-Pox . . . 78
Cretinism . . . 374
Croup 189
Cyanosis . . . 272
Cynanche Laryngea . 148
" Tonsillaris . . 281
" Trachealis . . 189

Degenerations— 15
" Calcareous . . 20
" Colloid . . . 19
" Fatty . . . 16
" Lardaceous . . 18
" Mucous . . 18
" of the Liver . . 325
" Pigmentary . . 21

PAGE
Delirium Tremens . . 372
Dementia 374
Dengue 87
Desquamative Nephritis 442
Diabetes Insipidus . 469
" Mellitus . . 157
Diabetes Cataract . . 159
Diaphoretics . . 498
Diarrhœa 299
Diets for various Diseases 496
Digestive Organs, Diseases of 279
Dilatation of the Heart . 266
" of the Stomach . 295
Diphtheria . . . 104
Diphtheritic Palsy . 107
Diseases, General . . 22
" of the Brain . . 343
" Organs of Digestion 279
" Kidneys . . 438
" Liver . . . 313
" Lungs . . . 179
" Mouth . . . 279
" Nervous System . 343
" Œsophagus . . 282
" Pancreas . . 339
" Peritoneum . . 309
" Skin . . . 481
" Spinal Cord . . 435
" Spleen . . . 339
" Stomach . . 285
" Supra-Renal Capsules . . . 480
" Urinary Organs . 438
Disseminated Cerebro-Spinal Sclerosis . 396
Diuretics . . . 498
Dropsy 10
" Acute Inflammatory 442
" after Scarlet Fever 84
" of the Brain . . 350
Dry Tetter . . . 492
Dysentery . . . 113
Dyspepsia . . . 285
Dysphagia . . . 283
Dysphonia Clericorum . 185

PAGE
Eccentric Cardiac Hypertrophy . . . 266
Echinocci . . . 330
Ecthyma . . . 490
Eczema . . . 484
Elephantiasis . . 494
Embolism . . . 8
Emetics . . . 499
Emphysema . . . 230
Emprosthotonos . . 412
Empyema . . . 215
Encephalitis . . . 343
Encephaloid Cancer . 176
Endocarditis . . . 249
" Ulcerative . . 249
English Cholera . . 98
Enteritis . . . 296
Epidemic Cholera . . 93
Epilepsy . . . 381
Epithelial Cancer . . 176
Epithelioma . . . 176
Equine Farcy . . 141
" Glanders . . 141
Erb's Palsy . . . 406
Eruptive Fevers . . 67
Erysipelas . . . 122
Erythema . . . 493
Exanthemata . . 493
Exophthalmic Goître . 276
Expectorants . . 499

Face, Paralysis of the . 426
Famine Fever . . 35
Farcy 141
Fatal Jaundice . . 328
Fatty Degeneration . 16
" Infiltration . . 21
Favus 487
Febricula . . . 32
Fever, Continued . . 22
" General Treatment of . . . 28
" Infantile Remittent 58
" Intermittent . 116
" Relapsing . . 35
" Remittent . . 121

PAGE
Fever, Rheumatic . . 145
" Simple . . . 32
" Symptomatic . 22
" Typhoid . . 48
" Typhus . . 41
" Yellow . . . 63
" Eruptive . . 67
Fish Skin . . . 492
Fits, Apoplectic . . 360
" Epileptic . . 381
Five Day Fever . . 32
Follicular Disease of the Pharynx . . 185
" Stomatitis . . 279
Frambœsia . . . 495

Gall Bladder, Inflammation of . . . 337
Gall Stones . . . 338
Gangrene of the Lung . 225
Gargles . . . 500
Gastric Ulcer . . 291
Gastritis, Acute . . 288
" Chronic . . 299
Gastrodynia . . . 286
General Diseases . . 22
" Paralysis of the Insane . . 378
Glanders . . . 141
Globus Hystericus . 410
Glosso-Labio-Laryngeal Paralysis . . 399
Goître, Exophthalmic . 276
Gonorrhœal Rheumatism 147
Gout 151
" Retrocedent . . 153
Gouty Kidney . . 450
Gooseberry Bug . . 487
Grey Tubercle . . 172
Grocers' Itch . . 482

Hæmatemesis . . 9
Hæmatinuria . . . 470
Hæmaturia . . . 471
Hæmophilia . . . 175
Hæmoptysis . . . 9

PAGE
Hæmorrhages of the Lung 242
Hay Asthma . . . 183
" Fever . . . 183
Heart, Anatomical Relations of . . 253
" Atrophy of the . 268
" Dilatation . . 267
" Fatty Degeneration 269
" Hypertrophy . 266
" Inflammation . 251
" Murmurs . . 255
" Sounds . . . 255
" Valvular Diseases of the . . 254
Heartburn . . . 286
Hemiplegia . . . 417
Hepatic Abscess . . 317
" Disease . . . 313
Hepatitis . . . 315
Herpes 484
Hodgkin's Disease . . 343
Humid Tetter . . 484
Hybrid of Measles and Scarlatina . . 90
Hydatid Tumours of the Liver . . . 330
Hydrocephalus . . 350
Hydronephrosis . . 475
Hydropericardium . . 254
Hydrophobia . . 136
Hydrothorax . . 220
Hyperæmia . . . 5
" Active . . . 5
" Passive . . 5
Hypertrophy . . 3
" of the Heart . . 266
Hysteria . . . 410

Icthyosis . . . 492
Idiocy 373
Idiopathic Erysipelas . 122
" Fevers . . . 22
" Tetanus . . 411
Ileus 303
Impetigo . . . 491
Indigestion . . . 285

PAGE
Infiltration, Calcareous . 20
„ Fatty . . . 21
„ Pigmentary . . 21
Inflammation of the Brain 344
„ Biliary Bladder . 337
„ Cæcum . . 298
„ Intestines . . 296
„ Liver . . . 315
„ Mouth . . . 279
„ Peritoneum . . 309
„ Stomach . . 288
Infantile Spinal Paralysis 404
Influenza . . . 103
Inhalations . . . 500
Insanity . . . 373
Insolation . . . 370
Interlobular Emphysema 230
Intermittent Fever . 116
Intestinal Obstruction . 303
„ Worms . . 305
Intestines, Inflammation of 296
Intussusception . . 303
Itch 489

Jaundice . . . 334

Keloid 495
Kidney, Diseases of . 438
„ Amyloid . . 455
„ Contracted . . 450
„ Fatty . . . 454
„ Gouty . . . 450
„ Lardaceous . . 455
„ Large White . . 453
„ Movable . . 474
„ Waxy . . . 455

Landry's Paralysis . 436
Laryngeal Phthisis . 399
Laryngismus Stridulus . 385
Laryngitis . . . 184
„ Chronic . . 187
Lead Colic . . . 301
„ Palsy . . . 301
Lepra 492

PAGE
Leptomeningitis . . 351
Leucocythæmia . . 341
Leukæmia . . . 341
Lichen 482
Liniments . . . 500
Liver, Abscess of the . 317
„ Acute Atrophy of . 328
„ Amyloid Degeneration of the . 325
„ Cancer of the . 326
„ Cirrhosis of the . 320
„ Diseases of the . 313
„ Fatty Degeneration of the . . 324
„ Hydatid, Tumours of the . . 330
„ Inflammation of the 315
„ Syphilitic Cirrhosis of the . . 323
Lobular Pneumonia . 221
Locked Jaw . . . 411
Locomotor Ataxy . . 392
Lotions . . . 500
Lung, Cancer of . . 241
„ Collapse of . . 204
„ Gangrene of . . 225
„ Inflammation of . 221
Lupus 494
Lymphadenoma . . 343
Lympho-Sarcoma . . 343

Malignant Cholera . 93
„ Scarlet Fever . 81
„ Small-Pox . . 70
Mania 375
Measles . . . 88
Medicinal Rashes . . 495
Medullary Cancer . . 176
Melancholia . . . 375
Ménière's Disease . . 401
Meningitis, Acute . 343
„ Cerebro-Spinal . 61
„ Spinal . . . 435
„ Tubercular . . 350
Mercurial Tremor . . 406
Migraine . . . 390

PAGE
Miliaria 485
Mitral Valve, Disease of 258
Molluscum 494
Morbilli 88
Morbus Addisonii . . 480
" Brightii . . 441
Mouth, Inflammation of 279
Movable Kidney . . 474
Mumps 112
Murmurs of Heart, Cause of 257
Myelites 435
Myocarditis 251

Narcotics 500
Nephritis 438
Nettle-Rash 493
Night Blindness . . 171
Noma 380
Nostrils, Inflammation of 183
Nyctalopia 171

Obstruction of the Bowels 303
Œdema of the Glottis . 184
" Lungs 212
Œsophagus, Diseases of 282
Opisthotonos . . . 412
Ozœna 184

Painters' Colic . . . 301
Pancreas, Disease of . 339
Papular Eruptions . 482
Paralysis of the Insane, General 378
Paraplegia 421
Paralytic Walk . . 419
Parasitic Animals . . 487
" Eruptions . . 487
Parotid, Inflammation of 112
Parotitis 112
Pemphigus 490
Pericarditis 244
Pericardium, Effusion into 244
" Inflammation of . 244
Peritoneum, Diseases of 309
Peritonitis 309

PAGE
Pertussis 110
Phthisis 233
Phlegmonous Erysipelas 122
Pityriasis 492
Plague 93
Plastic Bronchitis . . 209
Pleurisy 213
Pleurosthotonos . . 412
Plica Polonica . . 488
Pneumonia 221
" Treatment of . 227
Pneumothorax . . 233
Podagra 151
Poliomyelitis Acuta . 404
Pompholyx 490
Porrigo Decalvens . 487
" Favosa . . . 487
" Scutalata . . 487
Prescriptions . . 497
Progressive Muscular Atrophy . . . 402
Prurigo 482
Pseudo - Hypertrophic Paralysis . . 403
Psora 489
Psoriasis 492
Puerperal Fever . . 129
" Mania . . . 376
Pulmonary Consumption 233
" Emphysema . . 230
" Valves, Disease of 259
Purpura 169
Pustulæ 491
Pyelitis 438
Pyæmia 127
Pyrexia 22
Pyrosis 286
Pythogenic Fever . . 48

Quartan Ague . . 116
Quinsy 281
Quotidian Ague . . 116

Rabies 136
Ramollissement of Brain 349
Reaction of Degeneration 417

	PAGE
Red Gum	482
Red Softening of the Brain	349
Refrigerants	501
Relapsing Fever	35
Remittent Fever	121
Renal Colic	473
Rheumatic Fever	145
Rheumatism, Acute	145
" Chronic	147
" Gonorrhœal	147
" Muscular	147
" Synovial	147
Rheumatoid Arthritis	147
Rickets	167
Ringworm	487
Roseola	90
Rötheln	90
Rubeola	90
Rupia	491
Saccharine Diabetes	157
St Anthony's Fire	122
St Vitus' Dance	387
Salines	501
Sarcinæ Ventriculi	286
Scabies	489
Scarlatina	81
Scarlatinal Dropsy	84
Scarlet Fever	81
Scirrhus Cancer	176
Scrivener's Palsy	405
Scorbutus	170
Scrofula	172
Scurvy	170
Sedatives	500
Serous Apoplexy	360
Seven-Day Fever	35
Shingles	484
Skin, Diseases of	481
Small-Pox	70
Soft Cancer	176
Sounds of the Heart	254
" Reduplication of	255
Spasmodic Asthma	210
Spastic Paralysis	406
" Walk	419

	PAGE
Spinal Cord, Diseases of	435
Spleen Ague Cake	340
" Diseases of	339
Splenic Fever	142
Sputa in Bronchiectasis	243
" Bronchitis	243
" Broncho-Pneumonia	243
" Phthisis	243
" Pneumonia	243
Squamous Eruptions	492
Stimulants	502
Stomach, Cancer of the	293
Stomatitis	279
Strangulation of the Intestines	303
Structure of the Œsophagus	282
Structural Diseases of Heart	254
Sugar, Tests for	161
Summer Cholera	98
Sunstroke	370
Supra-Renal Capsules, Diseases of	480
Sycosis	487
Symptomatic Fever	22
Syphilis	131
Tænia Solium	306
Tape-Worms	306
Tertian Ague	116
Tests for Albumen	468
" Sugar in Urine	161
Tetanus	411
Thoracic Aorta, Aneurism of	277
Thoracic Auscultation	180
Thread-Worms	305
Thrombosis	6
Thrush	280
Tinea Decalvens	487
" Favosa	487
" Lupinosa	487
" Sycosis	487
" Tonsurans	487
Tonics	502

PAGE
Tonsillitis 281
Tooth-Rash 482
Tophi 153
Traumatic Tetanus . 411
Trichosis Furfuracea . 487
Tricuspid Valves, Disease of 259
Tubercle, Nature of . 172
Tuberculæ 494
Tubercular Meningitis . 350
  „ Phthisis . . 233
Tuberculosis 172
Tumours of the Brain . 367
Typhlitis 298
Typhoid Fever . . 48
Typhus Fever . . 41

Ulcerative Endocarditis . 249
Ulcer of the Stomach . 291
Uræmia 463
Urine, Deficiency of the Chlorides . . 467
Urine in Disease . . 465
  „ in Health . . 465
Urticaria 493

Vaccinia 78
Valvular Diseases of the Heart 254
Varicella 80

PAGE
Varieties of Gravel . 477
Variola 70
  „ Confluens . . 70
  „ Discreta . . 70
Varioloid 77
Venous Pulse . . 165
Vertigo Auditory . . 401
Vesicular Emphysema . 230
  „ Eruptions . . 483

Walks, Ataxic . . 419
  „ Paralytic . . 419
  „ Spastic . . . 419
Wasting Palsy . . 402
Water-Brash . . . 286
Water-Hammer Pulse . 259
Waxy Kidney . . 455
  „ Liver 325
  „ Spleen 340
White-Celled Blood . 341
Whooping-Cough . . 110
Worms, Intestinal . . 305

Yaws 495
Yellow Atrophy of the Liver . . . 328
  „ Fever 28
  „ Tubercle . . 172

Zona 484

WELLCOME LIBRARY INSTITUTE

MEDICAL WORKS

PUBLISHED BY

E. & S. LIVINGSTONE.

PUBLISHED ANNUALLY.

Crown 8vo., price 1s. 6d., by Post, 1s. 8d.

Edinburgh Medical School Calendar

AND

GUIDE TO STUDENTS

Containing all the information required for Graduation in Medicine and Science at the University, and the Conjoint Board of the Royal College of Physicians and Surgeons, Edinburgh, and the Faculty of Physicians and Surgeons, Glasgow (Triple Qualification); and also for the Dental Diploma; with List of Lecturers, Dispensaries, Students' Societies, etc., and

EXAMINATION QUESTIONS.

'It is full of the most useful information. It ought to be a most valuable work of reference for the medical student.'—*Edinburgh Medical Journal.*

'Much time and money may be saved by a careful perusal of this little book.'—*Medical Press and Circular.*

'The Edinburgh Medical School Calendar for 1887-88 maintains its reputation for comprehensiveness and accuracy of information.'—*Scotsman.*

E. & S. LIVINGSTONE, 15 TEVIOT PLACE.

Symington (Johnson, M.D., F.R.S.E., F.R.C.S.E.) The Topographical Anatomy of the Child. Illustrated by 14 Coloured Plates and 33 Woodcuts. Foolscap. £2, 2s. 1887.

M'Lachlan (John, M.B., C.M., B.Sc.) The Anatomy of Surgery—(Surgical Anatomy). Illustrated with 74 Useful Engravings. Fcap. 8vo. 10s. 6d. 1887.

Whitaker (J. R., B.A.) Anatomy of the Brain and Spinal Cord. Illustrated with 22 Plates (12 Coloured). Fcap. 8vo. 4s. 6d. 1887.

Johnstone (Alexander, F.G.S.)

Botany Notes. 2 Parts. Fcap. 8vo. 2s. each Part.
Part 1.—Histology and Physiology.
Part 2.—Systematic Morphology.

Zoology Notes. 2 Parts. Fcap. 8vo. 2s. each Part.
Part 1.—Invertebrata.
Part 2.—Vertebrata.

The above 'Notes' are specially adapted for Students preparing for Professional Examinations in Medicine and Science.

Catechism Series.

Materia Medica—Part 1. Organic. Part 2. Inorganic.
Pathology—Part 1. General. Part 2. Special.

Also, in Note-Book Form, Fcap. 4to, with Blank Pages for Notes, each Part, 2s. 6d.; or on Cards in Case, each Part, 2s. 6d.

Greville (H.L., F.I.C., F.C.S.) Students' Hand-Book of Chemistry. Fcap. 8vo. Illustrated. 6s. 1887.

Craig (Wm., M.D., M.C., F.R.S.E.) Manual of Materia Medica and Therapeutics. Fifth Edition, Revised and Enlarged. Fcap. 8vo. 6s. 1887.

Complete Catalogues Free on Application.

E. & S. LIVINGSTONE,
Booksellers and Publishers,
15 TEVIOT PLACE, EDINBURGH.